

**U. S. ENVIRONMENTAL PROTECTION AGENCY**  
**Washington, D.C. 20460**

OFFICE OF  
PREVENTION, PESTICIDES  
AND TOXIC SUBSTANCES

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**MEMORANDUM**

**SUBJECT:** EFED RED Chapter for **Ethyl Parathion**  
PC Code No. 57501 ; CAS No. 56-38-2  
DP Bar codes:

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This memo summarizes the attached EFED Environmental Risk Assessment for the ethyl parathion RED. It includes suggestions for labeling and mitigation measures and identifies gaps and uncertainties resulting from outstanding data requirements. The assessment identified the following major issues of concern:

- Ethyl parathion is very highly toxic to birds, fish, aquatic invertebrates and small mammals, and poses a high acute risk to birds, mammals, and aquatic invertebrates, as well as high reproductive and ecological chronic risk to birds.
- Ethyl parathion is very highly toxic to pollinating insects such as bees, and has a well documented history of bee-kill incidents.

### ***Use Characterization***

The environmental risk assessment is based on the following use information for ethyl parathion:

- Ethyl parathion is an organophosphate insecticide registered for use on 9 crops. Sunflower, sorghum, and corn account for about two-thirds of use annually.
- The maximum single application rate (1 lb. ai/acre) is for cotton and sorghum. Six seasonal applications are permissible at a minimum 7 day interval, for a maximum seasonal rate of 6 lb. ai/acre.

### ***Ecological Risk Characterization***

EFED believes that the available fate and effects data support a conclusion that the use of ethyl parathion poses a high risk to nontarget organisms in terrestrial and aquatic environments. The toxicological and exposure data suggest strongly that acute and chronic effects on birds, acute effects on bees, and acute effects on aquatic invertebrates are likely to occur as a result of ethyl parathion applications:

- Ethyl parathion is “very highly toxic” to birds, and RQs calculated for avian effects far exceed levels of concern and the high RQs suggest that any minor nuances with respect to exposure modeling will not greatly alter the conclusions regarding high potential acute risks to birds. Studies cited in this chapter indicate that a suite of effects occur with short exposure to ethyl parathion. These include direct mortality, chronic as well as acute sublethal effects such as reproductive effects from acute exposure. Studies with the similar but less toxic organophosphate methyl parathion suggest other possibilities such as changes in maternal care and viability of young birds, and increased susceptibility to predation.
- The aquatic RQs are calculated based on PRZM-EXAMS simulations, which have some potential to overestimate exposure levels. However, the resulting risk quotients for freshwater and estuarine/marine invertebrates in particular are so high that the aquatic LOCs would be exceeded with even an order-of-magnitude reduction in the RQs. The impact of potential overestimations may be more important for risk conclusions for fish, since the acute RQs for fish are within an order of magnitude of the LOCs, and the available incident data for effects in fish is limited.
- Extensive incident data compiled for ethyl parathion confirm adverse effects to both humans and terrestrial wildlife. There are extensive incident data from linking ethyl parathion to accidental and intentional poisoning of humans as reported in OPP’s Notice of Intent to Cancel. These poisonings include sublethal effects, and in some cases, mortality. While these human incidents occurred before the extensive mitigation measures

put in place in 1991, protective clothing and reentry intervals do not protect wildlife in a treated field.

Wildlife incident data link bird and mammal mortality to ethyl parathion use. These exposures have been associated with labeled uses, accidental exposures, and intentional misuses of ethyl parathion.

The uncertainty in the environmental fate database for the highly toxic degradate ethyl paraoxon may lead to some *underestimation* of avian and mammalian exposure to biologically active ethyl parathion residues. Degradation of parent to ethyl paraoxon on the surfaces of leaves and avian food items may result in additional exposure to toxic residues which can result contribute to acute and/or chronic effects to birds, mammals, and reptiles.

### ***Water Resources Assessment***

The water resource assessment, based on the known fate properties of ethyl parathion along with limited monitoring data, concludes:

- Ethyl parathion is not likely to move appreciably through the soil to ground water, except in areas where the ground water is particularly vulnerable sites (e.g. shallow depth to ground water, highly permeable soils with low sorption capacities).
- Ethyl parathion can be expected to move to surface water via runoff or spray drift. Ethyl parathion has been detected at low concentrations (< 0.14 ppb) in non-targeted surface-water monitoring programs, but these instances are rare, and isolated. There are no targeted monitoring data for ethyl parathion. Monitoring programs in the State of California and urban runoff studies indicate very few detections (< 2.5 ppb) of ethyl parathion. These monitoring data were taken before the imposition of mitigation requirements such as a 100 foot downwind buffer for aerial sprays.
- Estimated concentrations of ethyl parathion in surface-water and ground water sources of drinking water (DWECS) were based on PRZM-EXAMS simulations, due to inadequate direct drinking-water monitoring data. Estimated drinking water concentrations for HED were derived using model simulations of the maximum cotton use rates. The DWECS for surface water were 36.29 µg/L for acute risk and 0.30 µg/L for chronic risk. The DWECS for ground water is 1.21 µg/L.
- EFED believes, qualitatively, that ethyl parathion is not likely to pose a significant chronic risk to drinking water nationally. Non-targeted monitoring data over many years have yielded a low detection rate in both surface water and ground water.

A first-tier assessment of possible transport of the major degradate 4-nitrophenol (paranitrophenol) to ground water and surface water is included in this chapter. This degradate is toxic, but since it has a different mode of action than ethyl parathion and ethyl paraoxon, it is not included in HED's tolerance expression. Because Tier 1 environmental fate and transport modeling of ethyl paraoxon was not possible, it was assumed that the mass of ethyl paraoxon in drinking water cannot physically exceed the mass of ethyl parathion. Therefore, it was assumed that the concentration of ethyl paraoxon in drinking water cannot exceed the concentration of ethyl parathion.

There is high uncertainty in the results of this drinking water and aquatic assessment beyond that introduced by the screening models, because: 1) the lack of monitoring data for ethyl parathion and ethyl paraoxon in drinking water 2.) the inability to evaluate the fate and transport of ethyl paraoxon, a toxicologically important degradate, 3.) uncertainties inherent in the surface-water and ground-water models, and in the assumptions used in these simulations, and 4) the inability of the surface-water models to simulate a 100-foot buffer for estimates of runoff.

***Data Gaps***

***Environmental Fate:*** The following data requirements have not been fully satisfied:

- 162-1 Aerobic soil metabolism
- 162-3 Anaerobic aquatic metabolism
- 162-4 Aerobic aquatic metabolism
- 163-1 Leaching and adsorption/desorption (soils were autoclaved, need confirmatory data)
- 164-1 Terrestrial field dissipation
- 164-2 Aquatic field dissipation
- 165-4 Accumulation in Fish

In addition, a major data gap in the environmental fate assessment is the lack of fate and transport data for ethyl paraoxon. Although foliar dissipation studies are not routinely required, a complete environmental assessment for ethyl parathion and its degradates requires an understanding of the routes and rates of dissipation from foliage. This information is needed because ethyl parathion is applied to foliage.

***Ecological Effects:*** The ecological toxicity data base is complete except:

- 122-1(a) Seed Germination/Seedling Emergence,
- 122-1(b) Vegetative Vigor,
- 122-2 Aquatic Plant Growth.

EFED suggests that the following language be included on the appropriate labels.

**Statement to minimize the potential for surface water contamination for all end-use products:**

This chemical can contaminate surface water through aerial spray applications. Under some conditions, it may also have a high potential for runoff into surface water after application. These include poorly draining or wet soils with readily visible slopes toward adjacent surface waters, frequently flooded areas, areas overlaying extremely shallow ground water, areas with in-field canals or ditches that drain to surface water, areas not separated from adjacent surface waters with vegetated filter strips, and areas overlaying tile drainage systems that drain to surface water.

***Environmental Hazard Labeling - In Addition to Bee Precautionary Statements***

**Manufacturing Use Product**

“This pesticide is very highly toxic to aquatic organisms (invertebrates and fish) and wildlife. Do not discharge effluent containing this product into lakes, streams, ponds, estuaries, oceans, or public water unless this product is specifically identified and addressed in an NPDES permit. Do not discharge effluent containing this product to sewer systems without previously notifying the sewage treatment plant authority. For guidance, contact your State Water Board or Regional Office of the Environmental Protection Agency.”

**Emulsifiable Concentrate and Product with both Parathion and Methyl Parathion**

“This pesticide is very highly toxic to aquatic organisms (invertebrates and fish) and wildlife. Birds in treated areas may be incapacitated, have reduced number of offspring or killed. Shrimp and other aquatic organisms may be killed at recommended application rates. Do not apply directly to water, to areas where surface water is present or to intertidal areas below the mean high water mark, unless otherwise permitted in Directions for Use. Runoff and drift from target areas may be hazardous to aquatic organisms in adjacent aquatic sites. Do not apply when weather conditions favor drift or runoff from target areas. Do not contaminate water by cleaning of equipment or disposal of equipment washwaters.”

***Peer Reviewers***

This chapter was peer-reviewed by Dr. Ed Odenkirchen, Dr. Ed Fite, Mr. Brian Montague and Mr. Arnet Jones.

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## 1. ENVIRONMENTAL RISK ASSESSMENT

### a. Use Characterization

Ethyl parathion (*O,O*-diethyl *O*-p-nitrophenyl phosphorothioate) is a non-systemic, organophosphate insecticide with a broad spectrum of activity against agricultural insect pests. It has been used for agricultural pest control worldwide for over 40 years.

Cheminova Agro A/S is the sole producer of the technical ethyl parathion sold in the United States. Cheminova also produces parathion 8EC emulsifiable concentrate formulation, and a 6-3 EC mixture with their insecticide methyl parathion. Ethyl parathion is used on nine crops including sunflowers, sorghum, alfalfa, cotton, soybeans, barley, canola, and wheat. Sunflowers constitutes the highest use (23.43% of the ethyl parathion) among the nine crops.

The majority of ethyl parathion is used in the Central Plains including North Dakota, South Dakota, Nebraska, Kansas, Colorado, Oklahoma, New Mexico, and Texas. There are six other states with pockets of high use which include Arizona, Georgia, Alabama, Washington, Montana, and Delaware. The use of ethyl parathion according to state, ranked by usage data, is North Dakota, Kansas, Oklahoma, and Georgia. The use data indicates that no more than 600,000 lbs of ethyl parathion is used annually in the United States.

After several human poisoning incidents, an agreement was reached with Cheminova to limit the agricultural use of ethyl parathion. In 1992, EPA, in response to a request from Cheminova, announced the voluntary cancellation of all uses of parathion on fruit, nut, and vegetable crops. The only uses retained were those nine crops listed above.

Furthermore, a label restriction was required for ethyl parathion to reduce exposure to agricultural workers. This restriction mandates that ethyl parathion can only be applied by commercially certified aerial applicators and that treated crops may not be harvested by hand. Ethyl parathion is available only in closed handling systems for aerial applications. Ethyl parathion cannot be applied within 100 feet of buildings, public roads, or bodies of water. Also, it cannot be applied when wind speed exceeds 10 miles per hour. Ethyl parathion must be applied from nozzles located not more than 75% of the distance from the center of the aircraft to the wing tip or helicopter rotor tip.

The current label also contains a language warning of the hazards that this chemical can pose to human health: "Danger- Poisonous if swallowed, inhaled, or absorbed through skin or eyes". The label also contains warnings concerning fish, wildlife, and bees: "This pesticide is highly toxic to fish and wildlife" and "This product is extremely toxic to bees".

## b. Exposure Characterization

### i. Chemical Profile

1. Common Name: Ethyl Parathion
2. Composition: *O,O*-diethyl *O*-p-nitrophenyl phosphorothioate.
3. Class: Organophosphate.
4. Physical/Chemical properties:
  - Molecular formula:  $C_{10}H_{14}PSNO_5$
  - Molecular weight : 291.27
  - Physical state : Pale yellow liquid
  - Melting point :  $6.1^{\circ}C$
  - Vapor Pressure :  $3.5 \times 10^{-6}$  mm Hg
  - Water solubility : 24.0 ppm
  - Henry's constant :  $6.04 \times 10^{-7}$  atm-m<sup>3</sup>/mole
  - Log  $K_{ow}$  : 3.83

### ii. Environmental Fate

The environmental fate assessment for ethyl parathion is based on acceptable and supplemental data. All data requirements for ethyl parathion are fulfilled except for the Anaerobic Aquatic Metabolism (162-3), Aerobic Aquatic Metabolism (162-4), Batch Equilibrium Soil Column Leaching (163-1), Terrestrial Field Dissipation (164-1), Aquatic Field Dissipation (164-2), and Accumulation in Fish (165-4). A common data gap in these studies is associated with the analytical methods used for identification and quantification of ethyl parathion degradation products and the lack of confirmatory storage stability data. Since these problems may not be resolved through submission of additional data, new studies will be needed to confirm supplemental data used in the exposure assessments.

In addition to the Subdivision N data gaps, there are several other data gaps that limit a complete understanding of the environmental fate and transport of ethyl parathion. These data gaps are a lack of environmental fate and transport data for the highly toxic degradate ethyl paraoxon, and a lack of foliar interception and dissipation data for ethyl parathion and ethyl paraoxon. Although these data are not required according to Subdivision N guidelines, they would be useful for understanding the fate and transport of ethyl parathion and its degradate ethyl paraoxon, and therefore the potential for exposure to nontarget organisms.

Ethyl parathion degradation appears to be dependent on microbially mediated degradation, soil sorption, and to a lesser extent photodegradation. Ethyl parathion is stable to abiotic hydrolysis ( $t_{1/2} > 102$  days) in buffer solutions at pH 5, 7, and 9. Laboratory studies show that photodegradation of ethyl parathion also does not appear to be a major route of degradation. However, ethyl parathion may rapidly photodegrade ( $t_{1/2} = 4.4$  days) in irradiated aquatic

environments in the presence of photosensitizers. Photodegradation in the vapor phase can be another route of degradation of ethyl parathion. Minor phototransformation products (< 10% of applied) were 4-nitrophenol and ethyl paraoxon. Ethyl parathion is moderately persistent ( $t_{1/2}$ =58 days) in aerobic mineral soils. In contrast, it is metabolized rapidly ( $t_{1/2}$ =5.2 days) in aerobic aquatic environments. Ethyl parathion also degraded rapidly ( $t_{1/2}$ < 2 days) in anaerobic aquatic environments.

The degradation pathways of ethyl parathion are well understood for microorganisms, plants, and animals. The formation of paraoxon is dependent on oxidative desulfonation (cleavage of P=S bond to form P=O bond). This transformation can occur through photooxidation, chemical oxidation in the presence of dissolved  $O_2$  in water, oxidizing agents such chlorine or potassium permanganate, and enzyme mediated oxidation from oxidases. The predominate degradation (or detoxification) reaction of ethyl parathion is enzyme catalyzed hydrolysis of ethyl parathion to form p-nitrophenol and diethyl phosphothioate. Alkaline catalyzed abiotic hydrolysis, however, is another probable degradation (detoxification) pathway. The hydrolysis reaction entails cleavage of the P-O bond. Under reduced soil conditions, enzyme catalyzed reduction of the nitro group ( $-NO_2$ ) on the phenyl moiety can lead to the formation of aminophenols and aminoparathions.

Supplemental batch equilibrium studies suggest that ethyl parathion is expected to be relatively immobile ( $K_d$  = 9.1 to 25.3 ml/g) in mineral soils except in sand soils with low organic matter content. Open literature data indicate that the ethyl parathion sorption correlates with soil organic matter content; ethyl parathion had a mean  $K_{oc}$  of 2720 ml/g ( $K_d$  = 8.1 to 104 ml/g) in eight mineral soils. In contrast, ethyl paraoxon sorption appears to be highly correlated to clay content. Ethyl paraoxon had a range of  $K_d$ s from 0.82 to 49.4 ml/g. The degradate 4-nitrophenol also has low soil batch equilibrium ( $K_{oc}$  = 55 ml/g) coefficient. These data suggest that ethyl parathion may be less mobile in soil when compared with its degradates. Although ethyl parathion has a relatively low vapor pressure ( $3.5 \times 10^{-6}$  mm Hg) and Henry's Constant ( $6.04 \times 10^{-7}$  atm-m<sup>3</sup>/mole), it has been detected in the vapor phase or adsorbed onto particulate matter in air monitoring studies.

The major route of ethyl parathion dissipation in field studies appears to be dependent on degradation. The dissipation rate of ethyl parathion is variable ( $t_{1/2}$  = 3 and 32 days) for cotton fields in California and Missouri. In contrast, ethyl parathion rapidly dissipated from flood water with half-lives of less than 7 days in rice fields in Missouri and California. Neither ethyl parathion nor its degradate ethyl paraoxon were detected in soil samples in the aquatic field dissipation studies. While ethyl parathion does bioconcentrate in fish (BCF 430), depuration is rapid when source contaminant is removed (more than 98% reduction in residues after 14-days).

## 1. Degradation

### Hydrolysis studies (161-1) (Satisfied) (MRID # 40478701)

The abiotic hydrolysis study (MRID 40478701) provides acceptable data to fulfill the Hydrolysis (161-1) data requirement for ethyl parathion. These data indicate that ethyl parathion is stable to abiotic hydrolysis. No additional data are needed at this time.

Ring-labeled [<sup>14</sup>C]ethyl parathion (radiochemical purity 98.4%), at 6.8 to 8.1 ppm, hydrolyzed with half-lives of >30 days in sterile aqueous buffer solutions that were incubated at 25 ± 1 C in amber vials for 30 days. The half-lives of ethyl parathion were 133 days at pH 5, 247 and 356 days at pH 7 (HEPES and Tris buffers, respectively), and 102 days at pH 9. It is noteworthy that there is high degree of uncertainty on estimated abiotic hydrolysis half-lives because they were derived through data extrapolation. Several review articles, however, indicate that alkaline catalyzed hydrolysis is an important abiotic degradation pathway for ethyl parathion (Mulla, et al. 1981; Howard 1991).

### Photodegradation in water (161-2) (Satisfied) (MRID # 40644701, 42156001)

The photodegradation in water studies (MRID 40644701 and 42156001) provide acceptable data to fulfill the Photodegradation in Water (161-2) data requirement for ethyl parathion. These studies indicate that ethyl parathion photodegradation in water is highly dependent on the presence of sensitizers; photosensitizers enhance the photodegradation rate of ethyl parathion. No additional data are needed at this time.

Radiolabeled ethyl parathion, at 10 µg/ml, in sterile, pH 5 buffer solution had a half-life of 30 days when exposed to xenon light (MRID 40644701). In the presence of a photosensitizer (acetone), the half-life of ethyl parathion was 4.4 days. Ethyl parathion was stable ( $t_{1/2}$  >203 days) in dark control treatments. Definitive photodegradation products of ethyl parathion were 4-nitrophenol (8.4% of applied ethyl parathion), ethyl paraoxon (3.4% of applied) and CO<sub>2</sub> (12 to 14% of applied). The degradation products, S-phenyl or S-ethyl parathion, were tentatively detected at a cumulative concentration of 2.3% of applied. Two unidentified degradation products were detected at concentrations ranging from 11 to 14% of applied. An additional photodegradation study indicates that ethyl parathion photodegrades to form hydroquinone (14.2% of applied) and 4-nitrosophenol (5-6% of applied) (MRID 42156001). Also, one unidentified degradation product was detected (5 to 6% of applied). Similar degradation rates of ethyl parathion were found in the additional study. Open literature data of review articles indicate that ethyl parathion photodegradation is enhanced in the presence of natural photosensitizers (Howard, 1991). Major photodegradates of ethyl parathion are photodegradates are p-nitrophenol and paraoxon.

Photodegradation on soil (161-3) (Satisfied)  
(MRID # 40647702, 42025501)

The photodegradation on soil studies (MRID 40647702 and 42025501) provide acceptable data to fulfill the Photodegradation on Soil (161-3) data requirement for ethyl parathion. These studies indicate that ethyl parathion is relatively persistent to photodegradation on soil surfaces. No additional data are needed at this time.

Radiolabeled ethyl parathion, at 10  $\mu\text{g/g}$ , had a half-life of 73.1 days on a sandy loam soil when irradiated with xenon light for 28 days. Ethyl parathion had a half-life of 182 days in dark, control samples. Minor photodegradation products (<10% of applied) were ethyl paraoxon (3.4% of applied) and 4-nitrophenol (7% of applied). An additional unidentified degradation product (4.8% of applied) was also detected.

Photodegradation in air studies (161-4) (Satisfied)  
(MRID # 41126601, 42158201)

The photodegradation in air studies (MRID # 41126601, 42158201) provide acceptable data to fulfill the Photodegradation in Air (161-4) data requirement for ethyl parathion. These studies indicate that ethyl parathion is relatively persistent to vapor phase photodegradation in air. No additional data are needed at this time.

Radiolabeled ethyl parathion, at 464  $\mu\text{g/ml}$ , had a half-life of 61.4 days in air samples irradiated with a xenon sun lamp for 30 days. Ethyl parathion had a half-life of 1,117.2 days in dark controls. A minor photodegradation product (< 10% of applied) was ethyl paraoxon. Additionally, two unidentified products were detected at concentrations of < 10% of applied.

Open literature data indicate that photo-oxidation is a predominate degradation pathway for ethyl parathion to form ethyl paraoxon (Woodrow et al., 1983). Case studies indicate that ethyl parathion photodegradation is accelerated by the presence of an oxidant (ozone). Measured photodegradation rates of ethyl parathion in air are on the order of minutes.

## 2. Metabolism

Aerobic soil metabolism (162-1) (Supplemental)  
(MRID # 41187601, 42073101)

The aerobic soil metabolism studies (MRID 41187601, 42073101) provide upgradable supplemental data on the aerobic soil metabolism of ethyl parathion. The data are deemed as upgradable supplemental due to the lack of confirmatory degradate identification data and the lack



of formal storage stability data. These data can be upgraded with the submission of the following information:

- a) Sample storage conditions need to be specified.
- b) Storage stability data for paraoxon are needed.
- c) Kinetic analysis is required to assess the degradation rate of total (extractable plus nonextractable) ethyl parathion.
- d) The registrant should provide a complete assessment on the presence of ethyl paraoxon in the submitted studies.

Soil extractable ring-labeled [ $^{14}\text{C}$ ]ethyl parathion (radiochemical purity 99.5%), at 9.7 ppm, degraded with a half-life of 57.6 days in aerobic sandy loam soil incubated in the dark at 25 C and 75% of field capacity. The major degradate of ethyl parathion was identified as  $\text{CO}_2$ . Minor degradates (<10% of applied) were 4-nitrophenol, ethyl paraoxon, and O,O-bis(4-nitrophenyl) ethyl phosphate. Unextractable soil [ $^{14}\text{C}$ ]residues comprised a maximum of 49.1% of the applied at 92 days post-treatment and declined to 36.6% at 366 days. Non-extractable radiolabeled residues were tentatively identified as ethyl parathion.

An extensive review article indicates that ethyl parathion persistence can be highly variable (6 weeks to 16 years) in soils (Mulla et al., 1981). A possible factor controlling persistence may be related to application rate; higher application rates appeared to cause greater ethyl parathion persistence. The ethyl parathion degradation pathways are well understood for microorganisms, plants, and animals. In soils, microbial-mediated degradation is an important route of dissipation (Mulla et al, 1981 and Howard, 1991). The formation of paraoxon is dependent on oxidative desulfonation (cleavage of P=S bond to P=O bond). This transformation can occur through photooxidation, chemical oxidation in the presence of dissolved  $\text{O}_2$  in water, oxidizing agents such chlorine or potassium permanganate, and enzymatic mediated oxidation from oxidases. The predominate degradation (detoxification) reaction of ethyl parathion is enzyme catalyzed hydrolysis of the ester bond (P-O) which leads to the formation p-nitrophenol and diethyl phosphothioate. Alkaline catalyzed abiotic hydrolysis, however, is another probable degradation pathway (Schwarzenbach, et al. 1993). Under reduced soil conditions, enzyme catalyzed reduction of the nitro group can lead to the formation of aminophenols and aminoparathions.

Anaerobic aquatic metabolism studies (162-3) (Supplemental)  
(MRID # 41249801, 42451001)

The anaerobic aquatic metabolism studies (MRID 41249801 and 42451001) provide supplemental data on the anaerobic aquatic metabolism of ethyl parathion. The data are deemed supplemental because there are analytical discrepancies between the results of TLC and HPLC methods for ethyl parathion degradation products and inadequate verification of storage stability studies. Since these discrepancies may not be resolved through the submission of additional data, a new study is needed to confirm the rates and routes of degradation of ethyl parathion in anaerobic aquatic environments.

Radiolabeled ethyl parathion, at 9.8 ppm, degraded rapidly ( $t_{1/2} < 24$  hours) in flooded sandy loam soil when incubated in the dark at 19.2 to 27.8°C for 366 days (MRID 41249801 ). Radiolabelled ethyl parathion (radiochemical purity 99.5%), at 9.4 ppm, degraded with a registrant-calculated half-life of 49.2 hours in sandy loam soil that was incubated anaerobically (flooded plus nitrogen atmosphere) at approximately 25°C in the dark for 72 hours. 4-nitrophenol and *O,O*-bis(4-nitrophenyl)ethyl phosphate were identified in water and soil samples. Ethyl paraoxon was tentatively identified in soil samples through HPLC analysis. The quantities of these degradates cannot be assessed because of discrepancies between the results of TLC and HPLC methods. Cumulative  $^{14}\text{C}$  volatiles accounted for <0.1% of the applied radioactivity. Unextractable  $^{14}\text{C}$  residues in the soil comprised a maximum of 88.6% of applied radioactivity.

Aerobic aquatic metabolism studies (162-4) (Supplemental)  
(MRID # 41249802, 42476901)

The aerobic aquatic metabolism studies (MRID 41249802 and 42476901) provide supplemental data on the aerobic aquatic metabolism of ethyl parathion. The data are deemed supplemental because there are analytical discrepancies between the results of TLC and HPLC methods for ethyl parathion degradation products and inadequate verification of storage stability studies. Since these discrepancies may not be resolved through the submission of additional data, a new study is needed to confirm the rates and routes of degradation of ethyl parathion in aerobic aquatic environments.

Ring-labeled [ $^{14}\text{C}$ ]ethyl parathion (radiochemical purity 99.5%), at 10 ppm, degraded with a half-life of 5.2 days in flooded sandy loam soil when incubated aerobically in the dark at 25°C for 31 days. 4-nitrophenol and *O,O*-bis(4-nitrophenyl)ethyl phosphate were identified in soil extracts. Ethyl paraoxon was tentatively identified in soil and water extracts through HPLC analysis. Additionally, an unidentified degradate was detected in soil and water extracts. The quantities of these degradates cannot be assessed because of discrepancies between the results of TLC and HPLC methods. Unextractable [ $^{14}\text{C}$ ] residues comprised a maximum of 60.3% of the applied at 31 days posttreatment.

Open literature data suggest that ethyl parathion persistence in water is dependent on the pH, turbidity, and temperature (Mulla, 1981). There appears to be higher persistence in neutral and acidic environments and low temperatures. Ethyl parathion degradation will be accelerated in alkaline pH (Howard, 1991).

### 3. Mobility

Leaching and adsorption/desorption studies (163-1) (Supplemental)  
(MRID # 41076701)

This batch equilibrium study (MRID 41076701) provides supplemental data on the unaged portion of the Batch Equilibrium (163-1) data requirement for ethyl parathion. The data are deemed as supplemental because autoclaved soils were used in the studies. Since autoclaving may alter soil physicochemical properties and hence alter pesticide sorption affinities, EFED believes that additional batch equilibrium data on non-autoclaved soils are needed to confirm the supplemental data. Batch equilibrium or soil column leaching studies are also needed to assess the mobility of the toxicologically significant degradates, ethyl paraoxon and 4-nitrophenol.

Radiolabeled ethyl parathion (radiochemical purity 99.3%), in solution at approximately 0.12, 1.0, 2.0, 3.0 and 4.0  $\mu\text{g/ml}$ , had  $K_{\text{abs}}$  values of 1.7 ml/g ( $K_{\text{oc}} = 855 \text{ ml/g}$ ) for the sand, 25.3 ml/g ( $K_{\text{oc}} = 1580 \text{ ml/g}$ ) for the sandy loam, 11.3 ml/g ( $K_{\text{oc}} = 596$ ) for the silt loam, and 9.1 ml/g ( $K_{\text{oc}} = 232 \text{ ml/g}$ ) for the clay loam soils. Desorption coefficients ( $K_{\text{des}}$ ) for ethyl parathion were 1.85 ml/g for sand, 15.7 ml/g for clay loam, 14.3 ml/g for silt loam, and 10.3 ml/g for sandy loam soils.

Review articles indicate that ethyl parathion has a high sorption affinity to soil organic matter and activated carbon (Mulla, et al. 1981). Howard, 1991 reported batch equilibrium  $K_{\text{oc}}$ s for ethyl parathion ranging from 602 to 15,800 ml/g. Sanchez-Martin and Sanchez-Camazano, 1991 showed that ethyl parathion sorption is correlated with soil organic matter content. Ethyl parathion had a mean  $K_{\text{oc}}$  of 2720 ml/g ( $K_d = 8.1$  to 104 ml/g) in eight mineral soils. In contrast, ethyl paraoxon sorption appears to be highly correlated to clay content. Ethyl paraoxon had a range of  $K_d$ s from 0.82 to 49.4 ml/g.

#### Laboratory Volatility from Soil (163-2) (Satisfied) ( MRID # 40810902 )

The laboratory volatility studies (MRID 40810902) provide acceptable data to fulfill the Laboratory Volatility from Soil (163-2) data requirement for ethyl parathion. These studies indicate that ethyl parathion is not very volatile from soil surfaces. No additional data are needed at this time.

Radiolabeled ethyl parathion, 0.314  $\mu\text{g/g}$ , had a mean volatilization rate of  $5.3 \times 10^{-3} \text{ ug/cm}^2/\text{hr}$  from a sandy loam soil at 50% and 75% field capacity and air flow rates of 205 to 465 ml/min. The mean measured vapor pressure of ethyl parathion was  $3.5 \times 10^{-6} \text{ mm Hg}$ . In general, ethyl parathion had higher volatilization rates in soils with lower moisture content; otherwise, ethyl parathion was not related to air flow rates. Open literature data indicate that ethyl parathion has low volatility from soil surfaces (Howard, 1991). However, field monitoring data indicate that ethyl parathion can volatilize from leaf and soil surfaces (Woodrow et al., 1977; Majewski and Capel, 1995).

#### Field volatility (163-3) (Waived)

Field volatility studies have been waived because ethyl parathion exhibited low volatility in laboratory volatility studies. However, field monitoring data indicate that ethyl parathion can

volatilize from leaf and soil surfaces. Woodrow et. al., 1977 found that ethyl parathion (25W), applied at 2.2 kg/ha, on plum orchard caused detectable target-site air concentrations of ethyl parathion and ethyl paraoxon. Air concentrations of ethyl parathion ranged from 3554 ng/m<sup>3</sup> immediately posttreatment to 5.4 ng/m<sup>3</sup> at 21 days posttreatment. Air concentrations of ethyl paraoxon ranged from 302 ng/m<sup>3</sup> immediately posttreatment to 2.1 ng/m<sup>3</sup> at 2 days posttreatment. Majewski and Capel, 1995 found that ethyl parathion was detected in southeastern United States at concentrations ranging from 1.1 to 239 ng/m<sup>3</sup>. Also, ethyl paraoxon concentrations were detected in fog and air samples in CA. These detections were correlated with county wide parathion use in the San Joaquin and Imperial Valleys.

#### 4. Dissipation

Terrestrial field dissipation studies (164-1) (Supplemental)  
(41481101, 41187602, 41292500)

The terrestrial field dissipation studies (MRID # 41187601, 41187602 and 41292500) provide supplemental data on the dissipation of ethyl parathion (formulated as EC) when applied to cotton. The studies are deemed as supplemental data because 1.) the dissipation of 4-nitrophenol and O,O-bis (4-nitrophenyl) ethylphosphate were not addressed in the studies and 2.) storage stability studies indicate paraoxon may not be stable during soil sample storage. These deficiencies limit interpretation on the rates and routes of dissipation for ethyl parathion degradates. EFED believes that additional terrestrial field dissipation studies are needed to define the rate and routes of dissipation of ethyl parathion and its degradates under actual use conditions.

Ethyl parathion formulated as a 8 lb/gal EC, applied in six weekly treatments at 1.0 lb ai/A/application (total 6.0 lb ai/A), dissipated with a half-life of 32 days from the surface soil (0-to 4-inch) in a cotton field in Missouri (MRID #41481101). Ethyl parathion accumulated as a result of the repeated applications; the average concentration of ethyl parathion in the upper 4-inch depth steadily increased from 0.11 ppm immediately following the first application to 0.54 ppm immediately following the fourth application and was 0.15-0.17 ppm immediately following the fifth and sixth applications. Following the sixth application, ethyl parathion in the 0- to 4-inch depth was 0.12-0.13 ppm at 1-7 days, decreased to 0.092-0.094 ppm at 14-21 days, and was <0.05 ppm (not detected) by 56 days. In the 4- to 8-inch depth, ethyl parathion was detected only once, at 0.029 ppm immediately following the fourth application; it was not detected in any other soil sample taken below the 0- to 4-inch soil depth. The degradate ethyl paraoxon was not detected (<0.05 ppm) in any of the soil samples.

Ethyl parathion (8 lb/gal EC), applied in six weekly treatments at 1.0 lb ai/A/application (6.0 lb ai/A total), had field dissipation half-life of 3 days from surface soil (0- to 4-inch) in a cotton field in California (MRID # 41187602 and # 41292500). Ethyl parathion did not accumulate as a result of the repeated applications; the average concentration of ethyl parathion in the upper 4-inch soil depth was 0.39-0.47 ppm following the first four applications and was 0.26 ppm following the fifth and sixth applications. Following the sixth application, ethyl parathion decreased to 0.14 ppm at 1 day to <0.07 ppm by 7 days and was <0.05 ppm (not detected) by 14 days. Ethyl parathion was not detected in any soil sample taken below the 4-inch soil depth. The degrade ethyl paraoxon was not detected (<0.05 ppm) in any of the soil samples.

Although dissipation data are not required for pesticide registration, this type of data is important in addressing the terrestrial field dissipation behavior of foliar-applied pesticides. Willis and McDowell, 1987 reported that the mean foliar half-life for total (total and dislodgeable) ethyl parathion is 2.3 days (SD=2.96 days; n=44) from cotton, apples, cherry, orange, peach, alfalfa, carrot, citrus, cotton, endive, peas, quackgrass, chard, collards, leaf lettuce, and turnips. These data suggest that ethyl parathion is not persistent on foliar surfaces. However, the actual route of dissipation cannot be derived from the referenced data. Field monitoring data suggest ethyl parathion can volatilize from leaf and soil surfaces (Woodrow et al., 1977; Majewski and Capel, 1995). Woodrow, et al., 1977 found that ethyl parathion (25W), applied at 2.2 kg/ha, on plum orchard resulted in whole-leaf ethyl parathion concentrations of 147 µg/g immediately posttreatment which declined to 0.43 µg/g at 21 days posttreatment. Ethyl paraoxon had a maximum whole-leaf concentration of 1.9 µg/g at 5 days posttreatment which declined to 0.40 µg/g at 21 days posttreatment.

Aquatic field dissipation studies (164-2) (Supplemental)  
(MRID # 41481102, 41187603)

The aquatic field dissipation studies (MRID # 41481102, 41187603) provide upgradable, supplemental data on the aquatic field dissipation of ethyl parathion. The data are deemed as upgradable, supplemental because storage stability studies are needed to assess ethyl parathion stability in water samples. Because there are no current aquatic uses for ethyl parathion, this data requirement is not needed to support reregistration of ethyl parathion.

Ethyl parathion formulated as 8 lb/gal EC, applied as six weekly treatments at 0.188 lb ai/A/application or a total of 1.1 lb ai/A, had a field dissipation half-life of <7 days from flood water in rice paddies in Missouri and California (MRID# 41481102 and 41187603). The degradates ethyl paraoxon and 4-nitrophenol were not detected (<0.01 ppm) in any water samples. Ethyl parathion and ethyl paraoxon were not detected (<0.05 ppm) in any soil samples.

## 5. Accumulation

Laboratory studies of pesticide accumulation in fish (165-4) (Supplemental)

(MRID 40988101)

The fish accumulation study (MRID 40988101) provides supplemental data on the bioaccumulation of ethyl parathion in fish tissues. The data are deemed as supplemental because the identity and concentrations of radiolabeled residues in fish tissue and exposure water are needed. These data are needed to confirm the water concentration of ethyl parathion as well as to evaluate the ethyl parathion residues in fish tissues.

Radiolabeled ethyl parathion had BCFs in bluegill sunfish (Lepomis macrochirus) of 84X, 990X, and 430X for edible, non-edible, and whole fish tissues, respectively (MRID 40988101). After 14 days depuration, total residues decreased by >98% in edible and non-edible tissues.

## 2. SURFACE WATER ASSESSMENT FOR ETHYL PARATHION

### a. First-Tier Drinking-Water Assessment

The GENEEC model (version 1.2, May 3, 1995) was used to derive upper-bound estimates of the concentrations of ethyl parathion that might be found in surface water due to use on cotton or sorghum. The peak estimated environmental concentration (EEC) of ethyl parathion in surface water is 166  $\mu\text{g/l}$ . This was the value recommended to HED as the highly conservative Tier I estimate of *acute* drinking-water exposure for the human-health risk assessment. EFED recommended a highly conservative Tier I *chronic* drinking-water exposure estimate of 23  $\mu\text{g/l}$  (Table 3), based on the 56-day average GENEEC EEC. These estimates are based on the highest total annual use rate of 6.0 lbs ai/acre that is recommended for cotton and sorghum (i.e. 1.0 lbs a.i./acre  $\times$  6 applications). The input values for GENEEC are listed in Table 2.

<b>Table 2. GENEEC Input Parameters</b>			
<b>Input Variable</b>	<b>Input Value</b>	<b>Source</b>	<b>Status</b>
Chemical Name	Ethyl Parathion	EFED One-liner	N/A
Solubility	24.0 ppm	EFED One-liner	N/A
Hydrolysis $T_{1/2}$	Stable	MRID # 40478701	Acceptable
Photolysis $T_{1/2}$	30 days	MRID # 40644701	Acceptable
Aerobic Soil Metabolism $T_{1/2}$	174 days <sup>1</sup>	MRID #41187601	Acceptable
Aerobic Aquatic Metabolism $T_{1/2}$	5.2 days	MRID #41249802	Acceptable
$K_{oc}$	232 <sup>2</sup>	MRID # 41076701	Supplemental <sup>3</sup>
Application Rate	1.0 lb a.i./acre	Label (EPA Reg. # 4787-15)	N/A
Max. Number of Applications per year	6 <sup>4</sup>	Label (EPA Reg. # 4787-15)	N/A
Interval Between Applications	7 days	Label (EPA Reg. # 4787-15)	N/A

(1)  $58 \times 3 = 174$  days to represent upper 90th percentile prediction.

(2) The smallest  $K_{oc}$  value was used in order to produce the highest (most conservative) exposure value.

(3) This study was considered supplemental and did not satisfy the guidelines because it was conducted using autoclaved soil.

(4) six applications, 7 days apart, were used in order to produce the highest exposure value.

<b>Table 3. GENEEC Concentrations for Ethyl Parathion Use on Cotton and Sorghum</b>				
<b>APPLICATION METHOD</b>	<b>Peak EEC (<math>\mu\text{g/l}</math>)</b>	<b>4-day EEC (<math>\mu\text{g/l}</math>)</b>	<b>21-day EEC (<math>\mu\text{g/l}</math>)</b>	<b>56-day EEC (<math>\mu\text{g/l}</math>)</b>
Aerial Application	165.96	136.83	58.12	23.08

The GENEEC screening model provides upper-bound values on the concentrations that might be found in ecologically sensitive environments because of the use of a pesticide. It was designed to be simple to use and to only require data which is typically available early in the pesticide registration process. GENEEC is a single event model (one runoff event), but can account for spray-drift from multiple applications. GENEEC is hardwired to represent a 10-hectare field

immediately adjacent to a 1-hectare pond that is 2 meters deep with no outlet. The pond receives a spray drift event from each application plus one runoff event. The runoff event moves a maximum of 10% of the applied pesticide into the pond. This amount can be reduced due to degradation on the field and the effects of soil binding in the field. Spray drift is equal to 5% of the applied rate for aerial spray application.

GENEEC incorporates conservative assumptions to provide pesticide concentrations that can be appropriately used in screening calculations. Drinking water from surface water sources tends to come from bodies of water that are substantially larger than the 1-hectare pond simulated by the model. Furthermore, GENECC assumes that essentially the whole basin receives an application of the chemical. In virtually all cases, basins large enough to support a drinking water facility will contain some fraction of area that does not receive the chemical. Furthermore, there is always at least some flow (in a river) or turn over (in a reservoir or lake) of the water so the persistence of the chemical near the drinking-water facility is usually overestimated by GENECC.

If a risk assessment performed using GENECC results does not exceed the level of concern, then one can be reasonably confident that the risk will also be below the level of concern. However, since GENECC can substantially overestimate true drinking water concentrations, it is necessary to refine the GENECC estimate if the level of concern is exceeded.

#### b. Tier II Water Assessment

Since the EECs derived from first-tier GENECC simulations were above HED's drinking-water level of concern (DWLOC), Tier II EECs were calculated using PRZM 3.12 for simulating the agricultural field and EXAMS 2.97.5 for fate and transport in surface water. Each Tier II assessment simulated a single site that represents a high-end exposure scenario for the use of ethyl parathion on a particular crop. The meteorology and agricultural practices were simulated over multiple years (usually 36 years) such that the probability of an EEC occurring at that site could be estimated.

##### i. Details of Specific PRZM/EXAMS Scenario Input Parameters

Tier II PRZM/EXAMS simulations were performed using standard input files prepared for the following five crops: cotton, corn, alfalfa, sorghum, and soybeans. These five crops represent more than two-thirds of ethyl parathion usage in the United States. Standard input files were not available for the other four crops on the ethyl parathion label. Since results of these Tier II simulations still indicated exceedence of LOCs for most endpoints, EFED does not believe that PRZM/EXAMS simulations for the remaining four crops will alter the risk assessment.

The five input files were adapted to simulate the application of ethyl parathion for the respective crops and states represented in the standard scenarios. Chemical-specific input for ethyl parathion was derived to the greatest extent possible from the environmental fate database submitted to the EPA by registrant Cheminova. Application rates, numbers of applications, and application



intervals simulated were consistent with the maximum values requested by the registrants for establishing tolerances.

While the geographic sites used to build these scenarios may not represent areas of greatest ethyl parathion use, they are located in states where ethyl parathion is registered for these uses. Soils and weather data for these standard scenarios were extracted from the program PIRANHA, an input shell developed by ORD-Athens for the PRZM model. Emergence, maturation and harvest dates were also derived from PIRANHA, unless otherwise stated.

Further details are presented below:

## ii. Chemical-Specific Input

Persistence and mobility numbers used in the first-tier GENEEC simulations were also used for the Tier II assessment. Chemical specific input parameters for PRZM and EXAMS are summarized in Tables 9 and 10. Certain assumptions were made for chemical dissipation parameters included in PRZM 3.12 but not GENEEC:

1. PRZM input parameters representing aerobic soil metabolism (DSRATE and DWRATE) were developed from a single half-life measurement by multiplying that value by three ( $58 \times 3 = 174$  days) in accordance with current guidance. Subsoil layers were assumed to be aerobic as well, because the deepest soil layer simulated was only 150 cm deep. Consequently, the same parameter estimate was used for both surface and subsoil horizons.
2. Volatilization from the soil or foliage were not simulated (set to zero).
3. Dissipation pathways such as plant uptake and foliar degradation were not simulated;
4. Foliar wash off of  $0.5 \text{ cm}^{-1}$  rainfall was simulated;

PRZM and EXAMS require that degradation half-lives be converted into rate constants. The aerobic soil metabolism half-life of 174 days (as explained above) was converted to a daily rate constant for PRZM 3.12 by the equation  $\ln 2 / (T_{1/2})$ . The aerobic aquatic (input variable KBACW), anaerobic aquatic (KBACS), and photolysis (KDP) half-lives for EXAMS were converted to hourly rate constants using the formula  $\ln 2 / (T_{1/2} \times 24)$ . Hydrolysis was not considered in EXAMS because it is captured in the use of aquatic metabolism rate constants.

## iii. Crop-Specific Inputs

Cotton

This input file was adapted from a PRZM 2.3 scenario for cotton grown in Texas, dated June 2, 1998. Weather data from Major Land Resource Area (MLRA) H-77 is used for this scenario.

Crop	Emergence Date	Harvest Date	Application Dates	Application Method
Cotton	May 11	Sept. 12	June 1 - July 6	Aerial

This PRZM simulation reflects the maximum label rate (1.0 lb ai/a), number of applications (6/year) and application interval (7 days) sought by the registrants for ethyl parathion on cotton.

### Corn

This input file was adapted from a PRZM 2.3 scenario for corn grown on the Lynchburg loamy sand in Georgia, dated June 2, 1998. Thirty-six years (1948-83) of weather data from MLRA 133a are used for this simulation.

Crop	Emergence Date	Harvest Date	Application Dates	Application Method
Corn	April 11	Sept 12	July 1 to 26	Aerial

This PRZM simulation reflects the maximum label rate (0.75 lb ai/a), number of applications (6/year) and application interval (5 days) sought by the registrants for ethyl parathion on corn.

### Alfalfa

This input file was adapted from a PRZM 2.3 scenario for alfalfa grown on the Fury silty clay loam in Oregon, dated May 27, 1998. Thirty-six years (1948-83) of weather data from MLRA 23 are used for this simulation. Emergence, maturation and harvest dates were provided to EFED by Dr. Ben Simko, Extension Entomologist with the Malheur County, OR Cooperative Extension.

Crop	Planting Date	Harvest Date	Application Dates	Application Method
Alfalfa	March 22	September 7	April 1 to July 8	Aerial

This PRZM simulation reflects the maximum label rate (0.5 lb ai/a), number of applications and application interval (7 days) sought by the registrants for ethyl parathion on alfalfa. Ethyl

parathion is applied two times per cutting; this simulation incorporates this by having two weekly applications at the beginning of four successive months.

### Soybeans

This input file was adapted from a PRZM 2.3 scenario for soybeans grown on the Lynchburg loamy sand in Georgia, dated May 27, 1998. Thirty-six years (1948-83) of weather data from MLRA 133a are used for this simulation.

Crop	Emergence	Harvest Dates	Application Dates	Application Method
Soybeans	March 15 to 20	Mid-May to Aug 1 (max: Jun 10-Jul 20)	May 16 to June 19	Air Blast

This PRZM simulation reflects the maximum label rate (0.75 lb ai/a), number of applications (2/year) and application interval (7 days) sought by the registrants for ethyl parathion on soybeans.

### Sorghum

This input file was adapted from a PRZM 2.3 scenario for sorghum grown on a Loring silt loam in Kansas, dated December 30, 1997. Thirty-six years (1948-83) of weather data from MLRA 112 are used for this simulation.

Crop	Emergence	Harvest Date	Application Dates	Application Method
Sorghum	May 21	October 1	Aug 1 to Sept 5	Aerial

This PRZM simulation reflects the maximum label rate (1.0 lb ai/a), number of applications (6/year) and application interval (7 days) sought by the registrants for ethyl parathion on sorghum.

Tier II EECs derived from PRZM-EXAMS are presented in Table (9 ). Values provided to HED for human-health risk assessment are 36.29 ppb for acute exposure, and 0.30 ppb for chronic exposure. The scenarios simulated were chosen to represent sites expected to produce runoff greater than 90% of the sites where the appropriate crop is grown. Model inputs included the maximum application rates, maximum number of yearly applications, and the shortest recommended application interval. In spite of these conservative assumptions, the PRZM-EXAMS EECs are lower than those generated by Tier I GENEEC model runs.

**TABLE 9. ETHYL PARATHION ESTIMATED EECS CALCULATED USING PRZM/EXAMS MODELING**

<b>Crop</b>	<b>Alfalfa</b>	<b>Corn</b>	<b>Cotton</b>	<b>Sorghum</b>	<b>Soybeans</b>
<b>State</b>	Oregon	Georgia	Texas	Kansas	Georgia
<b>Application Rate (lb ai/acre)</b>	0.5	0.75	1.00	1.00	0.75
<b>Number of Applications</b>	8 (2 per cutting) 2 (1 cutting/season)	6	6	6	2
<b>Interval Between Applications (Days)</b>	7	5	7	7	7
<b>Acute (Peak) Conc.</b>	4.66 ppb 0.99ppb	39.80 ppb	54.65 ppb	60.91 ppb	13.94 ppb
<b>96 Hours Avg. Conc.</b>	4.17ppb 0.88 ppb	35.34 ppb	48.40 ppb	53.70 ppb	12.39 ppb
<b>21 Day Avg.Conc.</b>	2.90 ppb 0.65 ppb	25.69 ppb	33.10 ppb	37.41 ppb	8.55 ppb
<b>60 Day Avg.Conc.</b>	1.56ppb 0.37 ppb	13.43 ppb	20.16 ppb	22.34 ppb	4.66 ppb
<b>90 Day Avg. Conc.</b>	1.18 ppb 0.27 ppb	11.42 ppb	15.76 ppb	17.57ppb	3.36 ppb
<b>Chronic (Yearly) Conc.</b>	0.57 ppb 0.12 ppb	3.92 ppb	5.31 ppb	5.39 ppb	1.28 ppb

Chemical parameters used in the modeling of ethyl parathion are provided in Table (10).

**Table 10 . Exams Environmental Chemical Parameters**

Parameter	Value	Source
<b>Molecular Weight</b>	291.27	EFED One-liner
<b>Water Solubility</b>	24.0 ppm	EFED One-liner
<b>Henry's Law Constant</b>	6.04E-7 atm-m <sup>3</sup> /mol	EFED One-liner
<b>Soil Organic Carbon Partitioning Coefficient(K<sub>oc</sub>)</b>	816	MRID No. 41076701
<b>Vapor Pressure</b>	3.5E-6 mm Hg	MRID No. 40810902
<b>Hydrolysis Half-life</b>	Stable	MRID No. 40478701
<b>Aerobic Soil Half-life</b>	174 days	MRID No. 41187601
<b>Aerobic Aquatic Half-life</b>	15.6 days	MRID No. 41249802
<b>Anaerobic Aquatic Half-life</b>	147.6 hours	MRID No. 41249801

#### iv. Limitations of this Analysis

The use of simulation models to estimate possible drinking-water exposure introduces several degrees of uncertainty to a human health or ecological risk assessment. The greatest of these may be the conservative assumptions of the modeling that are intended to ensure the maximum protection for human health. The scenario simulated by both GENEEC and PRZM-EXAMS is a single 10-hectare field draining to a 1-hectare pond with no outlet. This represents a conservative assumption, since this scenario does not accurately reflect the dynamics in a watershed large enough to support a drinking water facility.

Additional assumptions ensure that the resulting Tier II EEC's are sufficiently conservative to protect human health and the environment:

- Sites simulated in Tier II modeling are chosen by best professional judgement to be among the most vulnerable for each crop to which the pesticide is applied.
- The 10-hectare field is assumed to be planted completely to the crop in question;

- Each individual application of the pesticide is assumed to occur over the 10 hectares within one day; and
- The application rates and timing for each crop are the maximum allowed on the product label.

A watershed large enough to support a drinking-water facility would rarely be treated uniformly with the same pesticide at the maximum label rate.

These conservative assumptions are intentionally chosen, in part, to account for other sources of uncertainty associated with the use of simulation models in risk assessment. The first of these is the quality of the input data used in the simulations, which is detailed to some extent above. In addition, the precipitation data used is limited to a maximum of 36 years, with no irrigation simulated in any year.

The assumption that direct deposit to the pond by spray drift would be 5% of the application rate for aerial applications might be particularly conservative for ethyl parathion. The 1992 agreement between EPA and Cheminova on the terms of registration for ethyl parathion included spray drift reduction measures which are reflected in PRZM/EXAMS EECs. These include a 100-foot no-application buffer from all water bodies, and the restriction that operating spray nozzles cannot be placed any further than 75% along the length of the spray boom. In order to address the impact of the 100-foot buffer strip on aquatic exposure, the aerial drift component (expressed as % of the applied rate) in the standard farm exposure assessment was modified to represent an average deposition from 100 ft to 200 ft downwind distance from the edge of the field. The average pesticide deposition in the pond was derived using numerical integration technique along a deposition curve, described by a three parameter first-order decay model ( $Y_0 + Y e^{-kt}$ ), derived from open literature aerial deposition data (Bird, 1996). Based on the spray drift assessment, the 100 foot buffer reduces the median drift from the 5% of applied default value to 2% of applied. It is not possible to simulate the effect of a 100-foot buffer on runoff from the treated field using PRZM and EXAMS. This fact adds additional uncertainty and conservativeness to the analysis.

Finally, the models themselves are a source of uncertainty in the assessments. While the models are some of the best environmental fate estimation tools available, they have limitations in their ability to represent some processes. Several of the algorithms (volume of runoff water, eroded sediment mass) are well validated and well understood, but no adequate validation has yet been made of PRZM 3.1 for the amount of pesticide transported in runoff events. Other limitations of the models used include the inability to handle spatial variability within the simulated 10-hectare field, a lack of crop-growth algorithms, and a simplistic soil water transport algorithm (the "tipping bucket" method).

Therefore, given these limitations, this Tier II EEC should be considered a reasonable upper bound estimate of the concentration that could be found in drinking water, and not a prediction of concentrations that would commonly be detected. Risk assessment using Tier II values can be used as refined screens to demonstrate that the risk to human health or the environment is below a level

of concern. When Tier II EEC values are above levels of concern, additional data or proactive mitigation measures may be necessary, depending on the magnitude of the LOC exceedence.

### 3. GROUND-WATER ASSESSMENT FOR ETHYL PARATHION

SCI-GROW is a screening level model developed by Dr. Michael Barrett to estimate the maximum groundwater concentration from the application of a pesticide to crops. SCI-GROW is based on the fate properties of the pesticide, the application rate, and the existing body of data from small-scale groundwater monitoring studies. The model assumes that the pesticide is applied at its maximum rate in areas where the groundwater is particularly vulnerable to contamination. In most cases, a considerable portion of any use area will have ground water that is less vulnerable to contamination than the areas used to derive the SCI-GROW estimates. As such, the estimated “maximum” concentration derived using SCI-GROW should be considered a high-end to bounding estimate of drinking-water exposure from a ground-water source. If the risk associated with this estimate is exceeded, either at the acute or chronic end-points, refinement of the exposure estimate will be necessary to better characterize actual exposures.

The input values for SCI-GROW are listed in Table 4. SCI-GROW predicts that the concentration of ethyl parathion in drinking water from ground sources is not likely to exceed 1.21  $\mu\text{g/l}$  (Table 5). SCI-GROW version 1.0 dated May 22, 1997 was used for the calculations.

Table 4. SCI-GROW Input parameters		
Input Variable	Input Value	Source
Chemical Name	Ethyl parathion	EFED One-liner
Aerobic Soil Metabolism $T_{1/2}$	174 days *	MRID # 41187601
$K_{oc}$	726 **	MRID # 41076701
Application Rate	1.0 lb a.i./acre	Label (EPA Reg. # 4787-15)
Max. # of Applications	6	Label (EPA Reg. # 4787-15)

\*  $58 \times 3 = 174$  days to represent upper 90th percentile prediction

\*\* Median Value

<b>Table 5. SCI-GROW Concentrations for Ethyl Parathion Use on Cotton and Sorghum</b>		
<b>APPLICATION METHOD</b>	<b>Total Annual Use Rate<sup>*</sup> (lbs a.i./acre)</b>	<b>SCI-GROW Acute and Chronic EEC (<math>\mu\text{g/l}</math>)</b>
Aerial Spray	6.0	1.21

\* The total annual use rate is equal to the application rate times the maximum number of applications allowed per year (i.e. 1.0 lbs/acre  $\times$  6 applications = 6.0 lbs/acre).

#### 4. MONITORING DATA

##### a. Surface Water Monitoring

Direct drinking-water data for ethyl parathion are not readily available, and it is not likely that many of such data have been collected. Public drinking-water supply systems must periodically analyze drinking water for contaminants that either: 1) have a Maximum Contaminant Level (MCL) established by the Office of Water, or 2) are included on the Unregulated Contaminant Monitoring List (UCML). Ethyl parathion does not have an established MCL, and is not included on the UCML. Therefore, public drinking water supply systems are unlikely to have analyzed for ethyl parathion.

A literature review in Howard, et al., 1991 cites papers from the 1970s and early 1980's which included analysis of ethyl parathion in drinking water. Other than a report of a single California drinking-water well which had a detection of 4.6 ppb ethyl parathion, the citations describe studies in which no ethyl parathion was found. However, the review does not describe necessary information such as detection limits, proximity to areas of ethyl parathion use or misuse, or whether the source water was surface water or ground water. In addition to these deficiencies, EFED notes that the cited studies do not reflect current use patterns and mitigation practices. These data are considered as anecdotal for this assessment.

Ethyl parathion has been included as an analyte in several national-scale surface-water monitoring studies since the mid-1960's. Ethyl parathion was detected in about 1% of the sites sampled (4 of 326) in these studies (Larson, et al., 1997). The maximum concentration reported is 2.5 ppb, from an urban runoff study in Fresno California (Oltmann, et al., 1985, cited in Larson, et al., 1997). This study had 51 detections in 86 samples collected from an area where parathion was "used heavily in agriculture in surrounding area". The fact that this study was undertaken in an area of heavy parathion use areas likely explains why the detection frequency and concentration was higher than in the other, nontargeted studies. The next highest concentration reported was from the USGS western stream survey of 1970-1971. Parathion was detected in 1% of the quarterly samples taken, with a maximum concentration of 0.16 ppb detected in a sample from the Sacramento River.



Ethyl parathion is among the analytes included in the United States Geological Survey's National Water Quality Assessment Program (NAWQA). Low levels of ethyl parathion were reported in preliminary results from samples collected from 1991-1995 from 20 major watersheds around the country. The maximum concentrations detected were as follows:

<b>Table 3: Surface Water Results, 1991-1995, USGS NAWQA Program</b>			
Type of Stream	# of Streams	# of Samples	Maximum Conc. (ppb)
Agricultural	37	1000	0.14
Urban	11	603	0.014
"Integrator"	14	555	ND

Parathion was also included and detected in the pilot study for the Kentucky River Basin, which took place between 1987 and 1990. The summary report for this study reports at least two detections of parathion at concentrations ranging between 0.08 and 0.11 ppb. The total number of samples and detections was not reported. In addition, ethyl parathion was detected in 4 of 30 streambed-sediment samples taken during the same time period. Because of the small number of samples taken, the authors did not attempt to associate the contamination with use on particular crops. Therefore, it is not known whether the detections were the result of use on crops still on the ethyl parathion label.

The concentrations in the studies cited above are below those predicted by the GENEEC screening model. It should be noted that the analytical recoveries for ethyl parathion in NAWQA is 58% (SD=8%). Other USGS data indicate that field spike of ethyl parathion in 1493 samples at 0.1 ug/L had medium recovery of 101% with a range from 9% to 220% [written communication from Jeff Martin, USGS (9/3/99)]. These data indicate that field recoveries appear to be reliable; however, there is enough variability to limit extensive quantitative interpretation of the monitoring data. The conservative assumptions used in the model for a first-tier assessment are expected to predict conservative drinking water concentrations. Additionally, the NAWQA program is not targeted directly for ethyl parathion and hence may yield lower concentrations. This study was designed to study the effects of agricultural runoff, but ethyl parathion is only one of a suite of many pesticides included in the water analyses. There is no guarantee of how well samples taken in this program correspond to times or locations of actual ethyl parathion use.

Ethyl parathion has been found at low concentrations in the San Joaquin-Tulare basins in agricultural drainage for many years. The USGS publication *Pesticides in Surface and Ground Water of the San Joaquin-Tulare Basins, California: Analysis of Available Data, 1966 Through 1992* details 57 samples taken from tile drains and other agricultural discharge which were analyzed for ethyl parathion. The maximum concentration detected in these samples, which were collected primarily by the California Department of Water Resources, was approximately 0.9 ppb. The report does not list the number of samples which resulted in detections of ethyl parathion, but the majority of these samples produced non-detects.

## b. Ground-Water Monitoring

Ethyl parathion has been detected in ground water, but these detections have been at low concentrations. The Pesticides in Ground Water Database (PGWDB) includes data from 3,529 wells, of which 3 showed positive detections of ethyl parathion. The highest ground-water concentration reported from these wells was 99 ppb, from a well in Georgia, a value which is unlikely to reflect non-point movement of ethyl parathion. The other detections, from Missouri and North Dakota, were of 0.2 and 0.02 ppb, respectively. Each of these detections occurred before mitigation agreements between Cheminova and EPA. The PGWDB does not include data for paraoxon.

Ethyl parathion was not detected in ground water in samples taken from the NAWQA program. As with the surface-water monitoring, the NAWQA ground-water monitoring study was not specifically targeted for times and areas of ethyl parathion use. Additionally, the analytical recovery for ethyl parathion in NAWQA is low [58% (SD=8%)] which limits extensive quantitative interpretation of the monitoring data.

Howard, 1991 lists two studies from the open literature which reported ethyl parathion detections in ground water. Ethyl parathion was reported at a concentration of 1 ppb in well water in Florida at 125-185 ft depth from agricultural source of contamination. It was also reported in a California ground-water aquifer at concentration ranging from 4 to 6  $\mu\text{g/L}$ . These citations predate current use and mitigation restrictions, and do not detail whether they were the result of agricultural use or point-source contamination. They will be considered as supplemental for the purposes of this assessment.

## 5. DRINKING-WATER ASSESSMENT FOR 4-NITROPHENOL

The degradate 4-nitrophenol, which is a degradate common to both ethyl parathion and methyl parathion, has been detected in drinking water. The EPA's National Pesticide Survey (NPS) reported that 4-nitrophenol was found in four samples, of which two were community water supply systems, and two private rural drinking-water wells. However, the study said that the analytical method used to detect 4-nitrophenol (GC/MS with electron capture) could not reliably quantify the concentration of the degradate in water.

It is important to note that 4-nitrophenol can be introduced into the environment by other pathways in addition to being a degradate of methyl parathion and ethyl parathion. This chemical is released in wastewater during the production of methyl parathion, ethyl parathion, and N-acetyl-p-aminophenol (pain-killer acetaminophen). 4-nitrophenol is also produced by photochemical reactions in the air connected with vehicular exhaust gas, and found on suspended particulate matter in the atmosphere.

Although 4-nitrophenol has been found in drinking water, the Health Effects Division has indicated that ethyl paraoxon is the only degradate of ethyl parathion included in the tolerance expression for ethyl parathion. Degradate 4-nitrophenol is toxic to humans, but it has a different mode of action and toxic endpoint than ethyl parathion and ethyl paraoxon. The endpoint of concern for 4-nitrophenol is children under 3 months old, due to concerns about methemoglobinemia. The EPA Office of Water has established one-day, ten-day and longer term Health Advisory levels (HA) for 4-nitrophenol of 800 ppb for a 10-kg child.

Therefore, some assessment of the potential of 4-nitrophenol to contaminate drinking water is warranted, in spite of the fact that it does not share a common mode of action with ethyl parathion and ethyl paraoxon. The uncertainty of such an assessment is significant, because EFED has not required that a full suite of environmental fate studies be performed for this chemical. Since 4-nitrophenol is produced in its own right as a fungicide used in the treatment of leather and cork insulation, EPA issued a RED for 4-nitrophenol in 1991. However, because 4-nitrophenol is only registered for indoor uses, the only environmental fate study that EFED requested be performed was the hydrolysis study. There is no indication that this study was ever submitted by registrant Monsanto.

The EFED chapter for 4-nitrophenol notes an aerobic soil metabolism half-life of 16 days, and a  $K_{oc}$  value of 214. No details are given on the sources of these data, nor the conditions under which these values were derived. A better source of peer-reviewed data comes from the National Library of Medicine, which has prepared a review of open literature studies on the chemical properties of 4-nitrophenol<sup>3</sup>. EFED performed a first-tier drinking water assessment for 4-nitrophenol using the data cited in that review:

<b>Table 6. GENEEC Environmental Fate Input Parameters for 4-Nitrophenol</b>		
<b>DATA INPUT</b>	<b>INPUT VALUE</b>	<b>SOURCE</b>
Effective Application Rate	0.52 lb ai/A (from methyl parathion) 0.13 lbs ai/A (from ethyl parathion)	Label rates adjusted* for % of degradate and difference in molecular weight
Maximum Number of Applications	10 (m-parathion) 6 (e-parathion)	Cheminova
Application Interval	3 days (methyl-parathion) 7 days (ethyl-parathion)	Cheminova
Batch Equilibrium (Koc)	55 ml/g	National Lib. Of Medicine
Aerobic Soil Metabolism	$t_{1/2} = 1.2$ days**	National Lib. Of Medicine
Solubility	16000 ppm	National Lib. Of Medicine
Aerobic Aquatic Metabolism	stable	N/A
Hydrolysis	stable	N/A
Photolysis	$t_{1/2} = 6.7$ days	National Lib. Of Medicine

\* Maximum application rate of parent compounds multiplied by the maximum amount of 4-nitrophenol detected (as % of applied parent) in any laboratory study submitted by the registrant multiplied by the molecular wt. Correction factor (i.e. M.wt.of 4-nitrophenol/M.wt of parent).

\*\* Half-life is from agricultural top soil experiment.

**Table 7. Surface Water Results for 4-Nitrophenol**

Use	App. Rate of Parent (lbs/acre)	Adjusted app. rate for degradate (lbs/acre)	# Apps/year	App. Int. (days)	GENEEC Peak EEC (ppb)	GENEEC 56 Day EEC (ppb)
Cotton	3.0 (MP)	0.52	10	3	42.42	40.66
Cotton	1.0 (EP)	0.13	6	7	8.02	7.69
<b>Total</b>	-----	-----	-----	-----	50.44	48.35

The values above include several conservative assumptions beyond those inherent in the GENEEC screening model itself:

- 1) The application rates used for 4-nitrophenol can be derived from the maximum rates at which parents methyl parathion and ethyl parathion are applied. These maximum rates were multiplied by the highest percentage of 4-nitrophenol found in any of the laboratory studies cited above and then multiplied by the molecular weight correction factor (i.e. M.wt.of 4-nitrophenol/M.wt of parent) . The maximum 4-nitrophenol derived from methyl parathion was 33%, from the anaerobic aquatic metabolism study. The maximum amount derived from ethyl parathion was 27%, from the aerobic aquatic metabolism study. Using these percentages to calculate an effective application rate assumes that other degradative processes are not occurring to degrade 4-nitrophenol as it is produced by the aquatic metabolism processes above. This is a *very* conservative assumption which should be considered when evaluating the results of this first-tier screen.
- 2) Since aerobic aquatic metabolism data is not readily available for 4-nitrophenol, this degradate was assumed to be stable to that process;
- 3) Since hydrolysis data is not readily available for 4-nitrophenol, this degradate was assumed to be stable to that process;
- 4) The additive risk from 4-nitrophenol derived from methyl parathion and ethyl parathion assumes that the uses of the parent compounds chosen are occurring in the same area for the GENEEC simulation. This is also quite a conservative assumption.
- 5) No other potential sources of 4-nitrophenol in drinking water are considered in this assessment. EFED is not aware of the magnitude of discharge of 4-nitrophenol in wastewater, or potential deposition in rainwater. It is possible that these sources might result in a more significant contamination of drinking water by 4-nitrophenol than the degradation of methyl parathion and ethyl parathion. No attempt to quantify the risk posed by other sources of 4-nitrophenol is attempted here.

In spite of the conservative assumption detailed above, the estimated concentrations of 4-nitrophenol in drinking water do not approach the 800 ppb HA for a 10-kg child. These values also

do not exceed OW's lifetime HA for a 70-kg adult of 60 ppb, but HED has indicated that adults are not an endpoint of concern for this chemical, in any case.

## 6. GROUND-WATER ASSESSMENT FOR 4-NITROPHENOL

Results of a SCI-GROW assessment for 4-nitrophenol are shown below. The assumptions made and chemical properties used to perform this assessment are the same as for the GENEEC run, with one exception. The aerobic soil metabolism half-life used in this assessment is 40 days, which was cited by the National Library of Medicine literature review as the half-life measured in subsoil samples. Using this half-life assumes that 4-nitrophenol quickly leaches to the subsoil, before degradation can occur in the top soil at the shorter half-life cited above.

<b>Table 7. Ground-water results for 4-Nitrophenol</b>				
<b>Crop</b>	<b>App. Rate of Parent (lbs/acre)</b>	<b>Adjusted app. Rate (lbs/acre)</b>	<b># Apps./Year</b>	<b>SCI-GROW Acute EEC (ppb)</b>
<b>Cotton</b>	3.0 (MP)	0.52	10	3.70
<b>Cotton</b>	1.0 (EP)	0.13	6	0.55
<b>Total</b>	-----	-----	-----	4.25

The PGWDB reports that 4-nitrophenol was detected in 3 of 263 wells sampled in Mississippi from 1982 to 1990, at concentrations ranging from 0.004 to 0.02 ppb. No detections were reported in 81 wells sampled in Washington in 1988. EFED recommends that a concentration of 4.25 ppb be used for a first-tier assessment of drinking water derived from a ground-water source.

## 7. ECOLOGICAL HAZARD ASSESSMENT

### a. Introduction

The toxicity of a pesticide is determined through laboratory testing of representative surrogate species. For instance, 2 surrogate species each are used in toxicity testing to represent all freshwater fish (>2000 species) and birds (>680 species) in the United States. Acute mammalian studies are usually performed using the Norway rat or the house mouse as surrogate species. Estuarine/marine testing is limited to a crustacean, mollusk, and fish. Reptiles and amphibians are not tested. Avian toxicity studies are used as surrogates for reptilian toxicity assessments. Fish toxicity studies are used as surrogates for amphibians, assuming that the tadpole stage has the same sensitivity as a fish.

The available acute toxicity data on the TGA I indicate that ethyl parathion is "very highly toxic" to "highly toxic" to birds ( $LD_{50} = 0.8989$  to  $16.9$  mg/kg;  $LC_{50} = 76$  to  $336$  ppm), "very highly toxic" to small mammals ( $LD_{50} = 2.52$  mg/kg, male rat), "highly toxic" to bees ( $LD_{50} = 0.175$  µg/bee), "very highly toxic" to "moderately toxic" to freshwater organisms ( $LC_{50} = 0.04$ - $3300$  ppb), and "highly toxic to moderately" to estuarine/marine organisms ( $LC_{50}$  or  $EC_{50} = 0.107$ - $1012$  ppb). Chronic toxicity studies established the following NOEC values:  $2.85$  ppm for avian species,  $1$  ppm for small mammals,  $0.002$  ppb for freshwater invertebrates,  $0.19$  ppb for estuarine/marine fish species, and  $0.0331$  ppb for estuarine/marine invertebrates.

#### b. Toxicity Mode of Action

Morgan (1982) provided the following description of the mode of action of organophosphate pesticides such as parathion:

Organophosphates poison insects and mammals primarily by phosphorylation of the acetylcholinesterase enzyme at nerve endings. The enzyme is critical to normal transmission of nerve impulses from nerve fibers to innervated tissues. Some critical proportion of the tissue enzyme mass must be inactivated by phosphorylation before symptoms and signs of poisoning are manifest. At sufficient dosage, loss of enzyme function allows accumulation of acetylcholine (the impulse-transmitter substance) at cholinergic neuroeffector junctions (muscarinic effects), and at skeletal myoneural junctions and in autonomic ganglia (nicotine effects). Organophosphates also impair nerve impulse transmission in the brain, causing disturbances in sensorium, motor function, behavior, and respiratory drive. Depression of respiration is the usual cause of death in organophosphate poisoning. Recovery depends ultimately on generation of new enzyme.

Organophosphates are efficiently absorbed by inhalation, ingestion, and skin penetration. To a degree, toxicity depends on the rate at which specific organophosphates are metabolized in the body (principally by hydrolysis in the liver), thus limiting the amount of pesticide available to attack acetylcholinesterase enzyme in other tissue.

Many organophosphates readily undergo conversion from -thions to -oxons (replacement of sulfur by oxygen). In general, -oxons are much more toxic than -thions. This conversion occurs in the environment under the influence of sunlight and in the body, mainly by the action of liver microsomes. Ultimately, both -oxons and -thions are inactivated by hydrolysis at the ester linkage, yielding alkyl phosphates and phenols which are readily excreted. The hydrolysis products present little toxic hazard.

Open literature studies on the ecological effects of methyl parathion are included in the risk assessment.

### c. Toxicity to Terrestrial Animals

#### i. Birds, Acute and Subacute

Acute oral toxicity studies using the technical grade of the active ingredient (TGAI) were performed to establish the acute avian toxicity of ethyl parathion. The preferred test species is either mallard duck (a waterfowl) or bobwhite quail (an upland gamebird). Results of the available tests are tabulated below.

#### Avian Acute Oral Toxicity

Species	% ai	LD50 (mg/kg)	Toxicity Category <sup>1</sup>	MRID No. Author/Year	Study Classification <sup>2</sup>
Mallard duck ( <i>Anas platyrhynchos</i> )	99.5	0.898	“very highly toxic”	00160000 Hudson/1970	Core
Mallard duck ( <i>Anas platyrhynchos</i> )	99.5	2.34	“Very highly toxic”	00160000 Hudson/1970	Core
Mallard duck ( <i>Anas platyrhynchos</i> )	99.5	1.44	“very highly toxic”	00160000 Hudson/1970	Core
Mallard duck ( <i>Anas platyrhynchos</i> )	98.7	1.9	“very highly toxic”	00160000 Hudson/1970	Core
Mallard duck ( <i>Anas platyrhynchos</i> )	98.76	2.13	“very highly toxic”	115198 Tucker/1970	Core
Japanese quail ( <i>Coturnix japonica</i> )	98.00	11.1	“highly toxic”	44323601 Rattner/1987	Supplemental
Japanese quail ( <i>Coturnix japonica</i> ) (acute heat)	98.00	6.8	“very highly toxic”	44323601 Rattner/1987	Supplemental
Japanese quail ( <i>Coturnix japonica</i> ) (chronic heat)	98.00	5.3	“very highly toxic”	44323601 Rattner/1987	Supplemental
Japanese quail ( <i>Coturnix japonica</i> )	0.98	11.5	“highly toxic”	44323601 Rattner/1987	Supplemental
Japanese quail ( <i>Coturnix japonica</i> ) (acute cold)	0.98	9.1	“very highly toxic”	44323601 Rattner/1987	Supplemental
Japanese quail ( <i>Coturnix japonica</i> ) (chronic cold)	0.98	7.6	“very highly toxic”	44323601 Rattner/1987	Supplemental
Fulvous whistling duck ( <i>Dendrocygna bicolor</i> )	98.7	0.125	“very highly toxic”	00160000 Hudson/1984	Supplemental
Red-winged Blackbird ( <i>Agelaius phoeniceus</i> )	NR	2.4	“very highly toxic”	05003191 Schafer/1984	Supplemental
House sparrow ( <i>Passer domesticus</i> )	98.8	3.4	“very highly toxic”	00160000 Hudson/1984	Supplemental
House sparrow ( <i>Passer domesticus</i> )	NR	1.3	“very highly toxic”	05003191 Schafer/1973	Supplemental



## Avian Acute Oral Toxicity

Species	% ai	LD50 (mg/kg)	Toxicity Category <sup>1</sup>	MRID No. Author/Year	Study Classification <sup>2</sup>
Quelea ( <i>Quelea quelea</i> )	NR	1.8	“very highly toxic”	5003191 Schafer/1973	Supplemental
Rock dove ( <i>Columba livia</i> )	98.7	2.52	“very highly toxic”	00160000 Hudson/1984	Supplemental
Gray partridge ( <i>Perdix perdix</i> )	98.7	16	“highly toxic”	00160000 Hudson/1984	Supplemental
Chukar ( <i>Alectoris graeca</i> )	98.7	24	“very highly toxic”	00160000 Hudson/1984	Supplemental
Japanese quail ( <i>Coturnix japonica</i> )	99.5	5.95	“very highly toxic”	00160000 Hudson/1984	Supplemental
Ring-necked pheasant ( <i>Fastens colchicum</i> )	99.5	12.4	“highly toxic”	00160000 Hudson/1984	Supplemental
California quail ( <i>Callipepla californica</i> )	99.5	16.9	“highly toxic”	00160000 Hudson/1984	Supplemental
Sharp-tailed grouse ( <i>Tympanuchus phasianellus</i> )	98	5.66	“very highly toxic”	00160000 Hudson/1984	Supplemental
Dermal					
Mallard duck ( <i>Anas platyrhynchos</i> )	NR	28.3 (percutaneous 24h feet exposed)	“highly toxic”	00160000 Hudson/1984	Supplemental
House sparrow ( <i>Passer domesticus</i> )	NR	1.8 (Dermal)	“very highly toxic”	05003191 Schafer/1984	Supplemental
Quelea ( <i>Quelea quelea</i> )	NR	1.8 (Dermal)	“very highly toxic”	05003191 Schafer/1984	Supplemental

<sup>1</sup> “Very highly toxic” is given to chemicals with LD<sub>50</sub>s less than 10 mg/kg and “highly toxic” designates chemicals whose LD<sub>50</sub> falls in a range between 10 to 50 mg/kg (Brooks,1973).

<sup>2</sup> Core (study satisfies guideline). Supplemental (study is scientifically sound, but does not fulfill the guideline)

Because the LD<sub>50</sub> falls in the range of <10 to 50 mg/kg, ethyl parathion is “very highly toxic” to “highly toxic” to avian species on an acute oral basis. Of the 12 species tested, the most sensitive species is the whistling fulvous duck based on the LD<sub>50</sub> of 0.125 mg/kg. The guideline (71-1) is fulfilled (MRIDNo.: 00160000). The dermal LD<sub>50</sub> for both the house sparrow and the quelea was 1.8 mg/kg. Brooks et al (1973) did not provide for dermal toxicity. However, because the dermal lethal dose is so very small, it is likely that under any scheme ethyl parathion dermal values would place it in the very highly toxic range.

Subacute dietary studies using the TGAI were performed to establish the toxicity of ethyl parathion to birds. The preferred test species are mallard duck and bobwhite quail. Results of these tests are tabulated below.

### Avian Subacute Dietary Toxicity

Species	% ai	5-Day LC50 (ppm) <sup>1</sup>	Toxicity Category <sup>2</sup>	MRID No. Author/Year	Study Classification
Mallard duck ( <i>Anas platyrhynchos</i> )	99.5	76(61-93)	"highly toxic"	00022923 Hill/1975	Core
Mallard duck ( <i>Anas platyrhynchos</i> )	99.5	275(183-373)	"highly toxic"	00022923 Hill/1975	Core
Northern bobwhite quail ( <i>Colinus virginianus</i> )	99.5	194(150-245)	"highly toxic"	00022923 Hill/1975	Core
Ring-necked pheasant ( <i>Phasianus colchicus</i> )	99.5	336(296-380)	"highly toxic"	00022923 Hill/1975	Supplemental
Japanese quail ( <i>Coturnix japonica</i> )	99.5	197(177-220)	"highly toxic"	00022923 Hill/1975	Supplemental

<sup>1</sup> Test organisms observed an additional 3 days while on untreated feed.

<sup>2</sup> "Highly toxic" is the designation for chemicals with LC50s between 50 and 500 ppm based on Brooks'(1973) classification scheme.

Because the LC<sub>50</sub> in a range between 50 to 500 ppm, Ethyl parathion is "highly toxic" to avian species on a subacute dietary basis. The guideline (71-2) is fulfilled (MRID # 00022923). Of the 4 species tested the most sensitive is the mallard duck.

#### ii. Birds, Chronic

Avian reproduction studies using the TGAI were performed for ethyl parathion because the following conditions were met: (1) birds may be subject to repeated or continuous exposure to the pesticide, especially preceding or during the breeding season,(2) information derived from mammalian reproduction studies indicates reproduction in terrestrial vertebrates may be adversely affected by the anticipated use of the product. The preferred test species are mallard duck and bobwhite quail. Results of these tests are tabulated below:

### Avian Reproduction

Species/ Study Duration	% ai	NOEC;LOEC (ppm)	LOEC Endpoints	MRID No. Author/Year	Study Classification
Mallard duck ( <i>Anas platyrhynchos</i> )	98	2.85/7.1	Eggs laid, eggs set, adult body weight, behavior, food consumption, hatchling body weight	41133101 Beavers/1989	Core
Northern bobwhite quail ( <i>Colinus virginianus</i> )	97	20/>20	(highest level tested) “... no apparent treatment related effects...”	41133102 Beavers/1989	Supplemental
Northern bobwhite quail ( <i>Colinus virginianus</i> )	Unknown	<25/25 <50	Time of oviposition Eggs laid	44329401 Rattner/1982	Supplemental
Gray partridge-Hungarian ( <i>Perdix perdix</i> )	Analytical grade	<8/8 (only 1 level tested)	Number dead in shells; early dead % hatch of fertile eggs	101170 Neill/1971	Supplemental
Mallard duck ( <i>Anas platyrhynchos</i> )	Technical	<10/10 (only 1 level tested)	Egg shell thickness	ESVII W1 Muller/1972	Supplemental

The mallard duck study (MRID 41133101) produced the lowest NOEC/LOEC, and will be used in the risk assessment. Therefore, an additional bobwhite quail study is not required, although the original study did not produce a NOEC for the bobwhite.

In addition to the above reproduction studies, Hudson et al. (1984;MRID 0160000) described 2, 60-day feeding studies, 1 with the fulvous whistling-ducks and 1 with gray partridges. The 2 fulvous whistling-ducks studied did not die on a diet with 1.5 ppm ethyl parathion. Ethyl parathion was lethal to 1 of the 2 gray partridges tested at 8 ppm. In 2, 30-day studies with repeated oral dosage (in mg/kg of bodyweight per day), the empirical minimum lethal dosage (30-day EMLD) for fulvous whistling-ducks and gray partridge were 0.01-0.02 mg/kg and 3.0-6.0 mg/kg, respectively.

### iii. Mammals, Acute and Chronic

Wild mammal testing is required on a case-by-case basis, depending on the results of lower tier laboratory mammalian studies, intended use pattern and pertinent environmental fate characteristics. In most cases, rat or mouse toxicity values obtained from the Agency's Health Effects Division (HED) substitute for wild mammal testing. Notice that in addition to the typical testing, Hudson et al.(1984) has provided LD<sub>50</sub>s for the domestic goat (*Capra hircus*) and mule deer (*Odocoiles hemionus*). These toxicity values are reported below.

## Mammalian Toxicity

Species/ Study Duration	% ai	Toxicity Value	Affected Endpoints	MRID No.
Laboratory rat Oral LD <sub>50</sub> 96 hours	Technical	♀2.52 ♂10.8 mg/kg	Mortality	243412
Laboratory rat Acute NOEC/LOEC	8E (76.8%)	8 mg/kg/16 mg/kg	Mortality	40814001
Laboratory rat Oral LD <sub>50</sub> 96 hours	Technical	♂2.7 ♀2.7 mg/kg	Mortality	243412
Domestic goat ( <i>Capra hircus</i> ) Oral LD <sub>50</sub>	98.7	28-56 mg/kg	Mortality	160000
Mule deer ( <i>Odocoileus hemionus</i> ) Oral LD <sub>50</sub>	99.5	22-44 mg/kg	Mortality	160000
Rat Inhalation LC50	NR	>1.3 mg/L♂ & ♀		003974
Rat Acute Dermal	76.8	LEL 16 mg/kg NOEL 8 mg/kg LEL 8 mg/kg NOEL 4 mg/kg	Mortality  Signs of toxicity	40814001
Rat Acute Dermal	76.8	LEL 0.68 mg/kg NOEL 0.45 mg/kg	Plasm ChE inhibiton	40814002
Norway rats (Wistar strain) Dietary LC50	Technical	126.3 ppm (103-186)	Mortality	43961101
Norway rats (Wistar strain) Dietary LC50	Technical	130.2 ppm (114-192)	Mortality	43961101
Mice 28 day- feeding	95.11	NOEL 50 ppm	100 ppm decreased body weight, tremors, hypoactivity 400 ppm animals died within 11 days	244841
Rat 3 month feeding	97.1 Technical	NOEL 0.04 mg/kg/day	Decreased ChE and EGR effects at 0.4 and 4 mg/kg/day	41834502

## Mammalian Toxicity

Species/ Study Duration	% ai	Toxicity Value	Affected Endpoints	MRID No.
Rat 3 month feeding	95.11	NOEL <2.5 ppm	Decreased RBC and plasma cholinesterase	244839
Dog 3 month feeding	Technical	Systemic NOEL >3.0 mg/kg/day (HDT) ChE NOEL <0.3 mg/kg/day (LDT)	Decreased RBC and plasma ChE activities	244843
Dog 1 year feeding	95.5 Pure	ChE NOEL 0.01 mg/kg Lowest Dose Tested.	(RBC, plasma and brain ChE were inhibited). Levels tested in beagles - 0, 0.01, 0.03 and 0.1 mg/kg/day	246639; 246642
Rat Reproduction	96.70 Technical	Fetotoxic NOEL 1.0 mg/kg Fetotoxic LEL = 4 mg/kg (with mortality and renal pelvis distention reported at 4 and 16 mg/kg).	Maternal toxic NOEL 1.0 mg/kg (Lowest Dose Tested) Maternal toxic LEL 4 mg/kg. (Decreased weight gain)	252087
Rat Developmental 2 generation	96.7 Technical	NOEL 1 ppm	F0 both sexes, cholinesterase activity was decreased in plasma and RBCs at 10 and 20 ppm. In the F1 pups reduced weight and weight gain was observed at the high dose. In the F1 adults plasma and cholinesterase activity was depressed, in males and females at 10 and 20 ppm. No effects were observed in the F2 pups.	41418501
Rat Feeding/ carcinogenic -2 year	96.7 Technical	NOEL 5 ppm	At 50 ppm showed tremors, abnormal gait, retinal degeneration, depression of all RBC values, depressed brain ChE, degeneration of sciatic nerve.	252702; 252703; 252704; 252705;
Rat Chronic/ carcinogenic feeding	96.70%	LEL(highest level tested) 32 ppm	Brain ChE depressed; gross retinal abnormalities, histopathology indicative of blindness and possible increase of blindness and mortality in females; decreased weight gain.	40644704

These studies show that ethyl parathion is toxic orally, dermally, reproductively, and chronically. The acute oral LD50 results indicate that ethyl parathion is (LD50 = 2.52 mg/kg) “very highly toxic” (<10 mg/kg) to small mammals on an acute oral basis (Brooks et al. 1973). The dermal studies although not analysed to provide an LD50 did show effects at the 8 mg/kg level. Therefore, the oral and dermal toxicities are similar. The rat reproduction studies show the fetotoxic LEL of 4 mg/kg and clinical chemistry effects at 10 ppm. Ethyl parathion toxicity increases with increases in the exposure period. For example, the lowest LC50 is for rats is 126.3 mg/kg. However, long term tests show that effects occur with exposure to concentrations as low as 1 ppm.

#### iv. Insects

Honey bee acute contact studies using the TGAI were performed for ethyl parathion because its use on alfalfa, cotton, and sunflowers will result in honey bee exposure. Results of this test are tabulated below.

#### Nontarget Insect Acute Contact Toxicity

Species	% ai	Results	Toxicity Category <sup>1</sup>	MRID No. Author/Year	Study Classification
Honey bee ( <i>Apis mellifera</i> )	Technical	0.175 µg/bee LD50	“highly toxic”	0036935 Atkins/1975	Core
Honey bee ( <i>Apis mellifera</i> )	Technical	100% Mortality@ 0.01% Solution	“highly toxic”	00078515 Harris/1970	Supplemental
Honey bee ( <i>Apis mellifera</i> )	4 lb E	Residues of 0.5 lbs a.i./A caused 100% mort. at 4 hr., 79% mort. at 1 day.	“highly toxic”	00091653 Johansen/1963	Core
Honey bee ( <i>Apis mellifera</i> ) Alkali bee ( <i>Nomia melanderi</i> ) Alfalfa Leafcutting bee ( <i>Megachile rotundata</i> )	4 lb E	At 0.5 lbs a.i./A 6-hr. residues highly toxic to all 3 species.	“highly toxic”	00060628 Johansen/1965	Core
Alkali bee ( <i>Nomia melanderi</i> )	Technical	LD50=1.29 µg/g	“highly toxic”	05015679 Moradeshaghi/1974	Supplemental
Eleven species of parasitic wasps and predaceous beetles	25 WP	At 0.5 lb a.i./A residues highly toxic after 4 days of exposure	“highly toxic”	05003978 Bartlett/1963	Supplemental
Sowbug ( <i>Asellus brevicaudus</i> )	98.7	LD50=2130 (1450-3120) µg/L	“highly toxic”	40094602 Johnson/1980	Core

<sup>1</sup> Based on Atkin (et al. 1981; MRID No.: 44038201) LD50 values less than 2 µg/bee are “highly toxic”. This is the highest toxicity category in this scheme of categories.

The results indicate that ethyl parathion is "highly toxic" to bees on acute contact basis. The guideline (141-1) is fulfilled (MRID No.0036935). The Johansen studies (00060628, 00091653) fulfill the guideline requirement for determination of residual toxicity of the formulated product to honeybees.

#### v. Terrestrial Field Testing

Field testing showed effects on young pheasants in a study where 60 9-week old penned birds were sprayed with 0.5 lbs ai/A ethyl parathion and given supplemental untreated food. Plasma and brain cholinesterase were depressed 59% and 30%, respectively, and ChE levels did not return to normal after 15 days. The authors concluded that re-exposure to parathion within this period could kill young birds. (Wolfe et al.,1971; MRID 44357801).

In another study 1 member of a nesting pair of laughing gulls that received a sublethal dose of ethyl parathion was compared with a group of non-dosed birds. After 2.5 days, birds dosed with 6 mg/kg parathion spent significantly less time incubating than non-dosed controls. By the third day, however, sharing of nest duties between pair members in the treated group had approached normal, suggesting some recovery from parathion intoxication. These findings suggest that sublethal exposure of nesting birds to an organophosphate insecticide, such as parathion, may decrease nest attentiveness and make the clutch more susceptible to predation or egg failure. The authors indicated that behavioral changes caused by sublethal OP exposure could be especially detrimental in avian species where only 1 pair member incubates or where both members are exposed in species sharing nest duties. (White, et al. 1983; MRID 44371709).

#### d. Toxicity to Freshwater Aquatic Animals

##### i. Freshwater Fish and Amphibians, Acute

Two freshwater fish toxicity studies using the TGAI were performed to establish the toxicity of Ethyl parathion to fish. The preferred test species are rainbow trout (a coldwater fish) and bluegill sunfish (a warmwater fish). Results of these tests are tabulated below.

#### Freshwater Fish Acute Toxicity

Species/  % ai	96-hour LC50 (ppb)	Toxicity Category	MRID No. Author/Year	Study Classification	
Bluegill sunfish ( <i>Lepomis macrochirus</i> )	98.7	18 (10-32)	“very highly toxic”	40098001 Mayer&Ellersieck/ 1986	Core
Bluegill sunfish ( <i>Lepomis macrochirus</i> )	100	24 (15-38)	“very highly toxic”	40094602 Johnson/1980	Core

## Freshwater Fish Acute Toxicity

Species/  	% ai	96-hour LC50 (ppb)	Toxicity Category	MRID No. Author/Year	Study Classification
Bluegill sunfish ( <i>Lepomis macrochirus</i> )	76.35	35 (17-52)	“very highly toxic”	40644710 Surprenant/1988	Core
Bluegill sunfish ( <i>Lepomis macrochirus</i> )	99	95 (NR)	“very highly txcic”	35796 Pickering/1962	Core
Bluegill sunfish ( <i>Lepomis macrochirus</i> )	99	710 (NR)	“ highly toxic”	57051 Henderson/1957	Core
Bluegill sunfish ( <i>Lepomis macrochirus</i> )	98.7	161 (75-346)	“ highly toxic”	40098001 Mayer&Ellersieck/ 1986	Core
Green sunfish ( <i>Lepomis cyanellus</i> )	98.7	395 (318-491)	“ highly toxic”	40098001 Mayer&Ellersieck/ 1986	Supplemental
Green sunfish ( <i>Lepomis cyanellus</i> )	Technical	207 (NR)	“ highly toxic”	44378608 Minchew/1969	Supplemental
Channel catfish ( <i>Ictalurus punctatus</i> )	98.7	2650 (2160-3260)	“moderately toxic”	40094602 Johnson/1980	Core
Channel catfish ( <i>Ictalurus punctatus</i> )	100	3300 (3090-3520)	“moderately toxic”	40094602 Johnson/1980	Core
Fathead minnow ( <i>Pimephales promelas</i> )	99	1300 (NR)	“moderately toxic”	35796 Pickering/1962	Core
Fathead minnow ( <i>Pimephales promelas</i> )	98.7	2350 (1760-3120)	“moderately toxic”	40094602 Johnson/1980	Core
Fathead minnow ( <i>Pimephales promelas</i> )	99	1400(NR)	“moderately toxic”	57051 Henderson/ 1957	Core
Fathead minnow ( <i>Pimephales promelas</i> )	100	330 (NR)	“highly toxic”	91881 Henderson/1959	Core
Mosquitofish ( <i>Gambusia affinis</i> )	99	350 (290-400)	“highly toxic”	44338801 Chambers/1974	Supplemental
Mosquitofish ( <i>Gambusia affinis</i> )	98.7	320 (156-647)	“highly toxic”	40094602 Johnson/1980	Core
Largemouth bass ( <i>Micropterus salmoides</i> )	98.7	620 (462-830)	“highly toxic”	40094602 Johnson/1980	Core



## Freshwater Fish Acute Toxicity

Species/  	% ai	96-hour LC50 (ppb)	Toxicity Category	MRID No. Author/Year	Study Classification
Goldfish ( <i>Carassius auratus</i> )	98.7	1830 (1350-2470)	“moderately toxic”	40098001 Mayer&Ellersieck/ 1986	Core
Golden shiner ( <i>Notemigonus crysoleucas</i> )	Tech	1895 (NR)	“moderately toxic”	44378608 Minchew/1969	Supplemental
Rainbow trout ( <i>Oncorhynchus mykiss</i> )	98.7	780 (370-1640)	“highly toxic”	40098001 Mayer&Ellersieck/ 1986	Supplemental
Rainbow trout ( <i>Oncorhynchus mykiss</i> ) static	98.7	1430 (964-2100)	“moderately toxic”	40094602	Core
Cutthroat trout ( <i>Oncorhynchus clarki</i> )	98.7	1560 (980-2470)	“moderately toxic”	40094602 Johnson/1980	Core
Lake trout ( <i>Salvelinus namaycush</i> )	98.7	1920 (1750-2100)	“moderately toxic”	40094602 Johnson/1980	Core
Chorus frog -tadpole ( <i>Pseudacris triseriata</i> )	98.7	1000 (700-14000)	“highly toxic”	40098001 Mayer&Ellersieck/ 1986	Core
Fowler’s toad-tadpole ( <i>Bufo woodhousei</i> )	98.7	>1000 (NR)	“moderately toxic or less”	40098001 Mayer&Ellersieck/ 1986	Core
Fathead Minnow ( <i>Pimephales promelas</i> )	Paraoxon 100%	250 (NR) hardwater	“highly toxic”	00091881 Henderson/ 1959	Core
Fathead Minnow ( <i>Pimephales promelas</i> )	Paraoxon 100%	330 (NR) softwater	“highly toxic”	00091881 Henderson/ 1959	Core

<sup>1</sup> Brooks (et al.,1973) toxicity classification indicates that LC50 values <0.1 ppm are “very highly toxic”, 0.1 to 1 ppm are “highly toxic and >1 to 10 ppm are “moderately toxic”.

The lowest LC<sub>50</sub> values fall in the range of less than 100 ppb. Ethyl parathion is "very highly toxic" to freshwater fish on an acute basis. The guideline (72-1) is fulfilled (MRID 40098001, 40647410). Of the 11 species tested, the bluegill is the most sensitive.

The available studies indicate that ethyl parathion is “highly toxic” to chorus frogs.(MRID No.: 40094602). At the present time there are no requirements for amphibian testing.

## ii. Freshwater Fish, Chronic

Freshwater fish early life-stage testing [72-4(a)] is required for ethyl parathion because the following criteria have been met:

- 1) It is likely to be a recurrent presence in water.
- 2) The fish LC50 is less than 1 mg/L.
- 3) Reproductive effects have been shown in both fish and invertebrates.
- 4) Half-life in water is 5.2 days.

No freshwater fish early life stage or life cycle data are available. However, two early life stage tests are available for the sheepshead minnow. Because of the similar acute toxicity between the freshwater bluegill sunfish and the marine spot, the most sensitive sheepshead endpoint NOEC 0.19 ppb was used in this risk assessment as a surrogate for a freshwater fish chronic endpoint. The results extremely, extremely high RQs, relative to LOCs, suggests that conduct of an early life stage test with a freshwater fish will not have significant effects on the outcome of the risk assessment.

## iii. Freshwater Invertebrates, Acute

Freshwater aquatic invertebrate toxicity tests using the TGAI were performed to establish the toxicity of ethyl parathion to invertebrates. The preferred test species is *Daphnia magna*. Results of these tests are tabulated below:

## Freshwater Invertebrate Acute Toxicity

Species	% ai	48-hour LC50/ EC50 (ppb)	Toxicity Category	MRID No. Author/Year	Study Classification
Crayfish ( <i>Orconectes nais</i> )	98.7	0.04 (NR)	“very highly toxic”	40094602 Johnson/1980	Supplemental
Crayfish ( <i>Procambarus sp.</i> )	98.7	<250(NR)	“highly toxic”	40094602 Johnson/1980	Supplemental
Waterflea ( <i>Simocephalus serrulatus</i> )	98.7	0.37 (0.23-0.57)	“very highly toxic”	40094602 Johnson/1980	Core
Waterflea ( <i>Daphnia pulex</i> )	98.7	0.6 (0.45-0.79)	“very highly toxic”	40094602 Johnson.1980	Core
Waterflea ( <i>Daphnia magna</i> )	76.35	3.0 (2.7-3.4)	“very highly toxic”	40644711 Surprenant/ 1988	Core
Waterflea ( <i>Daphnia magna</i> )		2	“very highly toxic”	43583501 Kühn/1998	Supplemental
Scud ( <i>Gammarus fasciatus</i> )	98.7	1.3 (0.6-1.9)	“very highly toxic”	40094602 Johnson/1980	Core
Scud ( <i>Gammarus lacustris</i> )	98.7	3.5 (2.6-4.8)	“very highly toxic”	40094602 Johnson/1980	Core
Scud ( <i>Gammarus fasciatus</i> )	98.7	4.5 (NR)	“very highly toxic”	40098001 Mayer& Ellersieck/ 1986	Supplemental
Fresh water shrimp ( <i>Palaemonetes kadiakensis</i> )	Technnical	7.1 (1.5-11.0)	“very highly toxic”	41237806 Naqvi/1970	Supplemental
Fresh water shrimp ( <i>Palaemonetes kadiakensis</i> )	Technnical	11.8(9.9-13.6)	“very highly toxic”	41237806 Naqvi/1970	Supplemental
Fresh water shrimp ( <i>Palaemonetes kadiakensis</i> )	Technnical	7.4(0.5-7.4)	“very highly toxic”	41237806 Naqvi/1970	Supplemental
Fresh water shrimp ( <i>Palaemonetes kadiakensis</i> )	Technnical	6.6(3.8-8.8)	“very highly toxic”	41237806 Naqvi/1970	Supplemental
Stonefly ( <i>Pteronarcys californica</i> )	98.7	5.4 (4.7-6.2)	“very highly toxic”	40094602 Johnson/1980	Core
Stonefly ( <i>Pteronarcella badia</i> )	98.7	4.2(3.4-5.2)	“very highly toxic”	40094602 Johnson/1980	Core
Stonefly ( <i>Claassenia sabulosa</i> )	98.7	1.5 (1.0-2.2)	“very highly toxic”	40094602 Johnson/1980	Core
Damselfly ( <i>Ischnura verticalis</i> )	98.7	0.64 (NR)	“very highly toxic”	40094602 Johnson/1980	Supplemental
Mayfly ( <i>Hexagenia bilineata</i> )	98.7	15(NR)	“very highly toxic”	40094602 Johnson/1980	Supplemental

<sup>1</sup> Brooks (et al., 1973) classification indicates the LC50 of 0.1 to 1 ppm are in the "highly toxic" range and those greater than 1 to 10 ppm are in the "moderately toxic" range.

Because most of the LC<sub>50</sub>/EC<sub>50</sub> values are less than 100 ppb, Ethyl parathion is "very highly toxic" to aquatic invertebrates on an acute basis. The guideline (72-2) is fulfilled (MRID 40094602, 40644711). These data suggest strongly that the use of ethyl parathion can lead to acute risk to freshwater invertebrates which could lead to significant effects to higher aquatic organisms which feed on the invertebrates. Because of their subtle nature, such aquatic effects would be difficult to detect. Of the 7 species tested, the crayfish (*Orconectes nais*) is the most sensitive.

#### iv. Freshwater Invertebrate, Chronic

A freshwater aquatic invertebrate life-cycle test using the TGAI was performed for ethyl parathion because it meets the following requirements: (1) it is expected to be transported to water from the intended use site, (2) it is intended for use such that its presence in water is likely to be continuous or recurrent, (3) the aquatic acute EC50 is less than 1 mg/l, or, (3) the EEC in water is equal to or greater than 0.01 of the acute EC50 value. Results of this test are tabulated below.

Freshwater Aquatic Invertebrate Life-Cycle Toxicity

Species/ Flow-through)	% ai	21-day NOEC/LOEC (ppb)	Endpoints Affected	MRID No. Author/Year	Study Classification
Waterflea ( <i>Daphnia magna</i> )		0.002/NR	parent mortality, reproduction rate, first offspring	43583501 Kühn/1989	Supplemental

Although these studies did not follow the guideline protocol, additional studies probably would not benefit risk assessment, because these studies show such extreme toxicity. Therefore, the requirement is reserved.

#### e. Toxicity to Estuarine and Marine Animals

##### i. Estuarine and Marine Fish, Acute

Acute toxicity testing with estuarine/marine fish using the TGAI was performed for ethyl parathion because it is expected to reach this environment when used in coastal counties. The preferred test species is sheepshead minnow. Results of these tests are tabulated below.

### Estuarine/Marine Fish Acute Toxicity

Species/(Static or Flow-through)	% ai	96-hour LC50 (ppb) (measured)	Toxicity Category	MRID No. Author/Year	Study Classification
Spot ( <i>Leiostomus xanthurus</i> )	99.6	18 (NR)	"very highly toxic"	40228401 Mayer/1986	Supplemental
Sheepshead Minnow ( <i>Cyprinodon variegatus</i> )	99.6	36 (NR)	"very highly toxic"	40228401 Mayer/1986	Supplemental
Striped mullet ( <i>Mugil cephalus</i> )	99.6	100 (NR)	"very highly toxic"	40228401 Mayer/1986	Supplemental

<sup>1</sup> Brooks (et al.,1973) classification indicates that LC50s less than 0.1 ppm or 100 ppb are "very highly toxic".

Ethyl parathion is "very highly toxic" to estuarine/marine fish on an acute basis. Although these studies are not core, an additional study would not be expected to benefit risk assessment. Therefore, the guideline (72-3a) is fulfilled (MRID 40228401) by these studies. Of the 3 species tested, the spot is the most sensitive.

#### ii. Estuarine and Marine Fish, Chronic

Because ethyl parathion's acute LC<sub>50</sub> is less than 1 ppm and applied several times per season, an estuarine/marine fish early life-stage toxicity test using the TGAI has provided.

### Estuarine/Marine Fish Early Life-Stage Toxicity Under Flow-through Conditions

Species/ Study Duration	% ai	NOEC;LOEC (ppb)	Endpoints Affected	MRID No. Author/Year	Study Classification
Sheepshead Minnow ( <i>Cyprinodon variegatus</i> )	98	0.19;0.37	weight and length	41543101 Surprenant/1988	Supplemental
Sheepshead Minnow ( <i>Cyprinodon variegatus</i> )	98.1	0.67;1.3	Hatchling survival	44347601 Sousa/1997	Core

The guideline requirements have been met for an estuarine/marine fish early life-stage study (MRID 44349401). Ethyl parathion is extremely toxic to estuarine/marine fish on a chronic basis.

### iii. Estuarine and Marine Invertebrates, Acute

Acute toxicity testing with estuarine/marine invertebrates using the TGAI was performed because ethyl parathion has the potential to reach the estuarine/marine environment when applied to crops grown in coastal counties. The preferred test species are mysid shrimp and eastern oyster. Results of these tests are tabulated below:

#### Estuarine/Marine Invertebrate Acute Toxicity

Species/Static or Flow-through	% ai.	96-hour LC50/EC50 (ppb)	Toxicity Category <sup>1</sup>	MRID No. Author/Year	Study Classification
Mysid ( <i>Mysidopsis bahia</i> )	76.35	0.107 (0.092-0.124)	“very highly toxic”	40644715 Surprenant/1988	Core
Mysid ( <i>Mysidopsis bahia</i> )	98	0.12 (0.11-0.14)	“very highly toxic”	40644714 Surprenant/1988	Core
Pink Shrimp ( <i>Penaeus duorarum</i> )	99.6	0.24 (NR)	“very highly toxic”	1237807 Lowe/1971	Supplemental
Brown shrimp ( <i>Penaeus aztecus</i> )	99.6	1.0 (NR)	“very highly toxic”	40228401 Mayer/1980	Supplemental
Grass shrimp ( <i>Palaemonetes pugio</i> )	99.6	2.8 (NR)	“very highly toxic”	40228401 Mayer/1984	Supplemental
Eastern oyster (shell deposition) ( <i>Crassostrea virginica</i> )	99	<1	N/A	41237807 Lowe/1971	Supplemental
Eastern oyster (shell deposition) ( <i>Crassostrea virginica</i> )	76.35	1012 (NR)	“moderately toxic”	40644717 Surprenant/1988	Supplemental
Eastern oyster (shell deposition) ( <i>Crassostrea virginica</i> )	99.6	>1000 (NR)	“highly toxic”	1237807 Lowe/1986	Supplemental

<sup>1</sup>Based on Brook's (et al. 1973) toxicity categories, chemicals with an LC50 less 0.1 ppm or 100 ppb and those between 0.1 and 1 ppm are “very highly toxic” and “highly toxic”, respectively.

The ethyl parathion LC<sub>50s</sub> for all but 2 of the above studies fall in the “very highly toxic” category for estuarine/marine invertebrates. The study under MRID No.: 1237807 indicates that ethyl parathion is “highly toxic”. The study under MRID No.: 41237807 was only tested at 1 ppb level showing no statistical differences. The guideline (72-3b) study is fulfilled by the mysid study (MRID No.:40644714). The mollusk study (72-3c) requirement is reserved based on the results of both MRID No.: 40644717 and 1237807 which show that relative to the mysid study it is much less sensitive. Of the the 5 species tested, the most sensitive is the mysid.

## iv. Estuarine and Marine Invertebrate, Chronic

## Estuarine/Marine Invertebrate Life-Cycle Toxicity

Species/(Static Renewal or Flow- through)	% ai	21-day NOEC; LOEC (ppb)	Endpoints Affected	MRID No. Author/Year	Study Classification
Mysid ( <i>Americamysis</i> <i>bahia</i> )	Technical	0.0031; 0.0052	Reproduction	40874401 Hoberg/1993	Supplemental
Sea Urchin ( <i>Pseudechinus</i> <i>magellanicus</i> ) Blastula	NR	EC50(CL) <sup>1</sup> 31.2(31.2- 52.0)	Abnormalities	44371708 Hernández/1990	Supplemental
Sea Urchin ( <i>Pseudechinus</i> <i>magellanicus</i> ) Gastrula	NR	EC50(CL) 34.6(27.2- 48.4)	Abnormalities	44371708 Hernández/1990	Supplemental
Sea Urchin ( <i>Pseudechinus</i> <i>magellanicus</i> ) Prism	NR	EC50(CL) 2.8 (1.0-6.5)	Abnormalities	44371708 Hernández/1990	Supplemental
Sea Urchin ( <i>Pseudechinus</i> <i>magellanicus</i> ) Pluteus	NR	EC50 (CL) 73.7 (46.7- 92.2)	Abnormalities	44371708 Hernández/1990	Supplemental

1. The results for these studies were provided as EC50 rather than NOEC/LOEC values.

The mysid study, which followed guideline protocol, did not provide the data necessary to adequately verify the NOEC. However, because of the extremely low NOEC the results of the risk analysis are not expected to change. Hence, another study (72-4) is not required.

Work with crabs shows effects can occur at lower concentrations with a longer exposure period. Rodriguez et al. (1992; MRID 443717107) determined a 96-hour LC50 and a 4-week LC50 for 2 species of crabs *Uca uruguayensis* and *Chasmagnathus granulata* collected in Argentina. For *Uca uruguayensis* only adults were tested while adults and juveniles were tested with *Chasmagnathus granulata*. The 96-hour LC50s for adult and juvenile *Chasmagnathus granulata* were 560 and 360 ppb, respectively, while the 4-week LC50s were much lower (4.35 and 10 ppb for adults and juveniles, respectively). Similarly, the *Uca uruguayensis* 96-hour and 4-week LC50s were 51 and 3.6 ppb, respectively. These studies suggest the potential for adverse chronic effects to invertebrates

in estuaries and marine areas, particularly in regions surrounding crops which can receive multiple applications of ethyl parathion.

#### f. Toxicity to Plants

##### i. Terrestrial

Currently, terrestrial plant testing is not required for pesticides other than herbicides except on a case-by-case basis (e.g., labeling bears phytotoxicity warnings incident data or literature that demonstrate phytotoxicity). Hartley et al. (1983) indicate that ethyl parathion is phytotoxic “to some ornamentals, cucurbits, sorghum, and some varieties of apple, pear, and tomato.” Due to the demonstrated phytotoxicity to some plants, Tier I terrestrial plants phytotoxicity tests are required (122-1, seedling emergence and vegetative vigor) in which the maximum registered label dosage is used. These studies are needed to determine the affect on endangered species.

##### ii. Aquatic Plants

Aquatic plant testing is required for the any pesticide with a phytotoxicity warning on product labeling, incident reports of phytotoxic effects, or other sources (such as published literature) of reported phytotoxicity. These studies are needed to determine the affect on endangered species.

The following species should be tested at Tier I: *Kirchneria subcapitata*, and *Lemna gibba*. Aquatic plant testing is required for parathion because of demonstrated phytotoxicity to plants.

#### 9. Risk Assessment

EFED uses an indexing method of risk assessment which considers exposure and toxicity components. Risk quotients (RQs) are calculated by dividing exposure estimates by toxicity values, both acute and chronic.

$$RQ = \text{EXPOSURE} / \text{TOXICITY}$$

RQs are then compared to OPP's levels of concern (LOCs). These LOCs are criteria used by OPP to indicate potential risk to nontarget organisms and the need to consider regulatory action. There are LOCs for the following risk presumption categories:

- (1) acute high - potential for acute risk is high and regulatory action may be warranted in addition to restricted use classification
- (2) acute restricted use - the potential for acute risk is high, but this may be mitigated through restricted use classification
- (3) acute endangered species - the potential for acute risk to endangered species is high and regulatory action may be warranted
- (4) chronic risk - the potential for chronic risk is high regulatory action may be warranted. (Currently, EFED does not perform assessments for chronic risk to plants, acute or chronic



risks to nontarget insects, or chronic risk from granular/bait formulations to mammalian or avian species.)

The toxicity test values (i.e., measurement endpoints) used in the acute and chronic RQs are derived from the results of required studies. Examples of toxicity values derived from the results of short-term laboratory studies that assess acute effects are:

- (1) LC<sub>50</sub> (fish and birds)
- (2) LD<sub>50</sub> (birds and mammals)
- (3) EC<sub>50</sub> (aquatic plants and aquatic invertebrates) and
- (4) EC25 (terrestrial plants).

Examples of toxicity test effect levels derived from the results of long-term laboratory studies that assess chronic effects are:

- (1) LOEC (Lowest Observed Effect Concentration)
- (2) NOEC (No Observed Effect Concentration)

The NOEC value is used as the toxicity test value in assessing chronic effects.

Risk presumptions, along with the corresponding RQs and LOCs, are tabulated below.

#### Risk Presumptions for Terrestrial Animals

Risk Presumption	RQ	LOC
Birds, Reptiles and Mammals		
Acute High Risk	EEC <sup>1</sup> /LC50 or LD50/sqft <sup>2</sup> or LD50/day <sup>3</sup>	0.5
Acute Restricted Use	EEC/LC50 or LD50/sqft or LD50/day (or LD50 < 50 mg/kg)	0.2
Acute Endangered Species	EEC/LC50 or LD50/sqft or LD50/day	0.1
Chronic Risk	EEC/NOEC	1

<sup>1</sup> abbreviation for Estimated Environmental Concentration (ppm) on avian/mammalian food items

<sup>2</sup>  $\frac{\text{mg/ft}^2}{\text{LD50} * \text{wt. of bird}}$     <sup>3</sup>  $\frac{\text{mg of toxicant consumed/day}}{\text{LD50} * \text{wt. of bird}}$

#### Risk Presumptions for Aquatic Animals

Risk Presumption	RQ	LOC
Acute High Risk	EEC <sup>1</sup> /LC50 or EC50	0.5
Acute Restricted Use	EEC/LC50 or EC50	0.1
Acute Endangered Species	EEC/LC50 or EC50	0.05
Chronic Risk	EEC/NOEC	1

<sup>1</sup> EEC = (ppm or ppb) in water

## Risk Presumptions for Plants

Risk Presumption	RQ	LOC
Terrestrial and Semi-Aquatic Plants		
Acute High Risk	EEC <sup>1</sup> /EC25	1
Acute Endangered Species	EEC/EC05 or NOEC	1
Aquatic Plants		
Acute High Risk	EEC <sup>2</sup> /EC50	1
Acute Endangered Species	EEC/EC05 or NOEC	1

<sup>1</sup> EEC = lbs ai/A<sup>2</sup> EEC = (ppb/ppm) in water**a. Risk Assessment to Nontarget Terrestrial Animals**

For pesticides applied as a liquid product, the estimated environmental concentrations (EECs) on food items following product application are compared to LC50 values to assess risk. The predicted maximum residues of a pesticide that may be expected to occur on selected avian or mammalian food items immediately following a direct single application at 1 lb ai/A are tabulated below.

Estimated Environmental Concentrations (EECs) on Avian and Mammalian Food Items (ppm) Following a Single Application at 1 lb ai/A)

Food Items	EEC (ppm) <sup>1</sup>
Short grass	240
Tall grass	110
Broadleaf/forage plants, and small insects	135
Fruits, pods, seeds, and large insects	15

<sup>1</sup> Maximum EEC are for a 1 lb ai/A application rate and are based on Fletcher *et al.* (1994).**i. Avian and Reptilian**Avian Acute Mortality Toxicity Assessment

The major uses of ethyl parathion are likely to result in bird and reptile deaths. In addition to mortality, a suite of sublethal effects has been documented in avian species. These include reproductive effects, health impacts for nesting birds and their young, damage to food resources, feeding and behavioral changes and greater vulnerability to predation and environmental stress.

***EFED concludes with a high level of confidence that ethyl parathion use poses significant acute and chronic risk to avian species.***

The acute and chronic RQs for broadcast applications of liquid products tabulated below are based on a mallard duck\* LC50 of 76 ppm and the mallard duck NOEC of 2.85 ppm.

Avian Acute and Chronic Risk Quotients for **Single** Application of Liquid Products  
(Broadcast)

Site(# App.)	App. Rate (lbs ai/A)	Food Items	EEC	Acute RQ (EEC/ LC50)	Acute LOC Exceedance	Repro- duction RQ (EEC/ NOEC)	Repro- duction LOC Exceed- ance
Alfalfa Canola (rapeseed)	0.5	Short grass	120.0	1.6	All	42.1	All
		Tall grass	55.0	0.7	All	19.3	All
		Broadleaf plants/Insects	67.5	0.9	All	23.7	All
		Seeds	7.5	0.1	Endangered Species	2.6	All
Barley Corn Soybeans Wheat	0.75	Short grass	180.0	2.4	All	63.2	All
		Tall grass	82.5	1.1	All	28.9	All
		Broadleaf plants/Insects	101.3	1.3	All	35.5	All
		Seeds	11.3	0.1	Endangered Species	4.0	All
Cotton Sorghum Sunflower	1	Short grass	240.0	3.2	All	84.2	All
		Tall grass	110.0	1.4	All	38.6	All
		Broadleaf plants/Insects	135.0	1.8	All	47.4	All
		Seeds	15.0	0.2	Restricted Use Endangered Species	5.3	All

\*Notice that the fulvous whistling duck is extremely sensitive to EP with an LD50 of 0.125 mg/kg versus 0.898 mg/kg for the next most sensitive species, the mallard duck. Based on the this these RQ may not protect this species. However, the lowest application rate and single application rate exceed all LOCs for the mallard.

Except for seeds, the single application scenario indicates that applications of ethyl parathion at all rates will result in RQs that exceed LOCs (endangered species, restricted use, and avian acute high risk) for both acute and reproduction toxicity values. The seed consumption RQ is below the high risk LOC for single applications of 0.75 lb ai/A and less. Notice that only a single application scenario was used for reproductive effects. The Corn Cluster Analysis (USEPA, 1996) reported that short

term exposure (Bennett et al.,1990) can cause similar effects as long term exposure with similar concentrations in the diet. This is illustrated in the case of the chemically similar pesticide methyl parathion. Bennett et al. (1990) exposed bobwhite quail to methyl parathion over both long-term (25 weeks) and short-term (4 days) exposure periods. Egg production in this species was statistically ( $p < 0.0001$ ) reduced at dietary concentrations 10 ppm for both exposure periods. Hence, if birds survive a short term exposure to ethyl parathion, they are likely to suffer reproductive effects.

#### Comparison of RQs Using LD50 and LC50

Ethyl parathion has been associated with many bird incidents (described later). Because the incidents are better explained by a single-dose poisoning, risk quotients based on the LD50 (single dose study) serve as a better indicator of potential risk than RQs based on the LC50 (5-day dietary study) as shown in the previous table (Stavola,1987).

The lowest LD50 is 1 mg/kg of bodyweight, hence a mallard-size bird need ingest, inhale, or absorb dermally only about 1 mg of ethyl parathion to face a 50% probability of mortality.

To compare the 2 types of exposure, RQs using the LD50 were compared to RQs using the LC50 according to the following calculations:

#### Information and Assumptions for Calculation of LD50/Day RQ:

1. Bird weight 1000 g for mallard duck
2. Formula to determine the weight of dry food per day

$$\text{Dry Food / day (g/day)} = 0.648Wt^{0.651}(\text{g}) \text{ (USEPA,1993)}$$

3. Dry food to wet food conversion = 1.8
4. RQ = mg/bird (lethal dose)/LD50

Formula:

$$\text{Dry Food (wt)Ingested/Day} * \text{Wet to Dry Weight} * \text{g to mg} * \frac{\text{EEC on Shortgrass/}}{\text{LD50}}$$

$$= \text{RQ LD50/Day}$$

$$0.648 (1000 \text{ g})^{0.651} * 1.8 (\text{dry to wet}) * 1000 \text{ mg/g} * (240/1,000,000) / 1 \text{ mg/kg}$$

$$= \text{RQ or 25.1 LD50 /Day}$$

Based on an application of 1.0 lbs a.i./A, the LD50 RQ for ethyl parathion is 25.1 while the RQ calculated from the LC50 is 3.2. Because ethyl parathion clearly causes effects with a single dose,

EFED believes that the LC50 EEC risk quotients in the table above significantly *underestimate* acute risk to birds and the LD50 values are more representative of potential risk.

### Avian Exposure Assessment

Birds sprayed with ethyl parathion can be poisoned by ingestion during preening, absorption through the skin of their feet, or by inhalation of spray particles. The RQs above do not take inhalation and dermal exposure into account, and an inhalation LC50 has not been determined for ethyl parathion. In addition, in dry conditions up to 30% of the parathion can be converted to paraoxon, which is 40 to 50 times more dermally toxic and 5 times more easily dermally absorbed than parathion. (USEPA, 1991).

Dermal toxicity tests suggest that birds in the vicinity of spray applications of parathion are likely to be affected by dermal exposure. The dermal LD50 value for the rat (21 mg/kg) is similar to the mallard duck percutaneous LD50 value of 28.3 mg/kg, which is based on direct exposure of their feet. The rat is used as the mammalian model for human exposure in dermal toxicity tests, and humans have been poisoned by dermal adsorption of ethyl parathion. Similarly, direct exposure to the skin under the wing resulted in dermal LD50 values for the passerines, quelea and house sparrow of 1.8 mg/kg. Based on these dermal LD50s, a house sparrow (28g) only needs a dose of 50  $\mu$ g of ethyl parathion to reach the LD50.

Based on similar effects seen in humans, Stavola (1987) suggests that inhalation and dermal exposure to ethyl parathion by birds will likely cause adverse affects. For example, people have been poisoned by inhalation or dermal exposure to ethyl parathion in spite of protective clothing. As a result, crops such as orchards which would require people to be near a treated field or to come in contact with treated foliage during harvest have been removed from the label. In addition, reentry restrictions have been established to keep flaggers and other field workers out of fields during treatment. Although such measures were effective for reducing the risk to humans, wildlife is still found in and around treated fields where people are not allowed, and these animals are likely to suffer adverse effects.

Multiple applications will increase the risk by increasing the opportunity for detrimental exposure (though it should be noted that this risk assessment does not include RQs for multiple applications). For instance, the label allows 6 applications to cotton and sorghum at 7-day intervals. By extending the duration of toxicity in a treated field, multiple applications can poison not only birds present at the time of each application, but intermittent visitors for weeks thereafter. Also, ChE levels may still be depressed from a previous application when birds are exposed again by a repeated application. Duration of toxicity may also be extended by degradation of ethyl parathion to paraoxon. Ethyl parathion intoxication can cause damaging effects in this population, even after residue levels fall below lethal levels. Birds that survive exposure to ethyl parathion can suffer behavioral changes that could disrupt mating, for instance, and prevent breeding. In addition, intoxication can also lead to greater susceptibility to predation.

Birds are likely to be in fields at the times of application as many of the crops to which ethyl parathion is applied are known to provide cover or serve as roosting or nesting sites. The opportunity for

exposure to toxic residues can extend beyond the time of application because: 1) multiple applications allowed for most crops can potentially lead to accumulation of ethyl parathion residues on foliage, and 2) degradation of ethyl parathion on foliage can produce the more toxic degradate paraoxon. Stavola (1987) cited studies indicating possible exposure to ethyl parathion and paraoxon up to 75 days following application in arid climates and up to 2 days in moister climates.

Human incidents have shown a persistence of toxicity with ethyl parathion which can cause poisoning long after application. In 1 carefully documented case, 4 workers became ill picking grapefruit 48 days after the application due to the build up of toxic paraoxon residue in the dust and on surfaces (Maddy, et al., 1985). A separate analysis by California Department of Food and Agriculture revealed 45 incidents in California from 1949 to 1986 involved multiple cases of poisoning due to entry at 1 site. Parathion was in 58 percent of these incidents, where the median time for worker reentry was 22 days. In contrast, the median time for other pesticides responsible for multiple cases of worker-reentry poisoning was only 1 day (Blondell, J. per comm., 1998 and California Department of Food and Agriculture, 1987). Based on multiple applications, persistence and formation of paraoxon, and human incident data, chronic exposure is likely to occur and can lead to reproductive and other sublethal effects in birds.

In addition, birds living along the edges of treated fields are also subject to dermal and inhalation exposure through spray drift. While a 100-foot buffer has been established from property lines, the buffer can be waived with written permission. EFED hopes to better assess the effectiveness of specific buffer zones to reduce spray drift in the near future with the spray drift model AgDRIFT.

Young birds are more likely to be exposed to ethyl parathion sprays than adults, due to lesser mobility and a greater area of exposed skin. Young birds hatched and reared along the edges of fields or among crops such as alfalfa will be directly exposed to spring and summer applications of ethyl parathion, and will not yet be able to fly from the path of the spray. Young songbirds, which lack feathers, will directly absorb residues through bare skin over much of their body. Nestlings further from a field can also absorb the chemical through direct contact with their parents' bodies when they return from foraging in treated fields.

#### Avian Incidents and Evidence of Acute Effects

A total of 52 incidents involving avian species are documented in the Ecological Incidents Database (see Appendix A). Analysis of the incidents confirms detrimental toxic effects with exposure to ethyl parathion in the field. The body of incidents, however, is not capable of demonstrating reproductive and other sublethal ecological effects that are likely to occur as a result of ethyl parathion use. It is very likely that the reported incidents greatly underestimate the acute and chronic effects that occur as a result of ethyl parathion use.

There are at least 6 incidents known in which geese were killed after grazing on winter wheat. One such incident from 1981 involved 1500 geese and 100 ducks. A 1993 incident involved 53 geese, 12 mallards and 4 teal which died after ingesting winter wheat and corn treated with ethyl parathion. A

1992 application to winter wheat led to the death of 3 prairie chickens. These incidents support the RQs that indicate high acute risk for birds at current label rates.

Many other incidents of birds killed by oral exposure to ethyl parathion have been reported. Many of these incidents, which include both seed-eating and insectivorous species, were found to have ethyl parathion residues in their gastrointestinal tracts. Some of the incidents included in the appendix were apparently the result of intentional misuse of ethyl parathion. One such intentional bird-kill in a rye field from 1982 affected over 3000 birds, which provides some insight to the number of birds that might be exposed by visiting treated fields. In most cases reported, not only were pest bird species affected but desirable species as well.

Other incidents were apparently caused by inhalation or dermal contact with ethyl parathion. In 1 incident, 6 swallows nesting above a doorway were killed by a mist of ethyl parathion and methyl parathion that the homeowner saw approach through trees on the edge of her property. Another 1990 incident involved 15 to 20 purple martins which died after application of ethyl parathion to a small grain field 200 yards away. Spray drift from an aerial application to wheat reached a chicken house 0.4 miles away and killed 649 chickens in the building. Eight domestic turkeys were killed in a garden by aerial drift of parathion from a tobacco field.

The spray drift mitigation measures included in the 1991 agreement may reduce drift to adjacent properties, but ethyl parathion is still expected to contaminate trees in wind breaks, fence rows, edge and wood lots that are near treated fields. Hawks and other predators can be expected to hunt from trees near fields, as the transition zones from field to trees provide excellent habit for their prey. Migratory red-tailed hawks have been poisoned in the past by delayed dormant orchard treatments, and may still be affected by inadvertant treatment of nearby trees.

#### Evidence of Indirect Poisonings

Bioconcentration can increase the likelihood of exposure and acute effects. In 1 study, mallard ducklings fed 5% of their bodyweight of tadpoles exposed to 1 ppm ethyl parathion died. In a similar experiment, 1 of 4 American kestrels fed cricket frogs exposed to 10 ppm ethyl parathion died in less than 3 hours after consuming 5 frogs (Fleming et al.1982). Amphibians can tolerate ethyl parathion turning them into toxic baits for sensitive birds (and reptiles).

#### **Reproductive and Sublethal Effects**

Because there is evidence of reproductive effects from a single exposure, RQs for reproduction were calculated from a single application. The single application scenario for reproductive effects shows that RQs range from 2.6 to 42.1 for the lowest application rate of 0.5 lbs/A. The highest rate, 1 lb ai/A, provided a range of 5.3 to 84.2. Multiple application scenarios will result in even higher RQs.

It is important to note that the damage done by sublethal effects, e.g., reduced number of eggs laid, reduced hatchling survival, increased susceptibility to predation due to lethargy or other behavioral

anomalies, is very difficult to detect. In short, reproductive and sublethal effects result in survival of fewer animals, hence, sublethal effects are as detrimental to populations as acute lethal effects.

As reported in the Corn Insecticide Cluster Analysis (1996), OP compounds may cause reproductive effects with short period of exposure. Several studies have shown that reproductive effects can occur in avian species exposed to OP compounds in the diet for 8 to 21 days (Bennett et al. 1991; Bennett and Bennett, 1990; Stromberg, 1981; Stromberg, 1986; Rattner et al., 1982). Further, Bennett et al. (1990) found that except for the number of adult mortalities, all effects observed in a long-term OP insecticide exposure test (25 weeks) were also observed in the short-term OP insecticide exposure test (10 days).

Environmental changes are likely to increase the sensitivity of birds to parathion. Rattner et al. (1982; MRID 44342003) found that the tolerance to cold may be reduced following ingestion of parathion. A bird that is inactive due to an abnormal intolerance to cold weather may not be able to forage sufficiently to survive or evade predators. These types of effects would most likely go unnoticed without a study designed to specifically look for these effects.

#### Endocrine Disruption

Ethyl parathion can have an adverse effect on reproduction. In mallards, for example, a concentration of 2.85 ppm led to significant reductions in eggs laid, eggs set, adult body weight, food consumption, and hatchling body weight compared to controls. Other studies with northern bobwhite quail and Hungarian gray partridge showed effects at 20 and 8 ppm. An acute exposure Rattner et al. (1982; MRID 44342003) reported that body weight, egg production, follicular development, and plasma lutenizing hormone (LH) and progesterone concentrations were reduced in birds receiving 100 ppm parathion compared with other groups (statistically significant at  $P < 0.05$ ). In another study where Japanese quail were dosed directly, LH was examined. At 4, 8, and 24 hours, LH was significantly decreased ( $P < 0.05$ ) at a dose of 5 mg/kg. (Rattner et al., 1986). These data suggest that ethyl parathion has endocrine disrupting properties.

#### Effects on Avian Food Supply

Ethyl parathion's very high toxicity to aquatic and terrestrial insects may have effects on birds by killing nontarget invertebrates and reducing food supply (USDI, 1951; Martin et al. 1951). Grue et al. (1988; MRID 443570802) reported on the effects of reduced food supply on ducks in the prairie-pothole region of the U.S., a region of ethyl parathion use. The study confirmed the dependence of ducklings and egg-laying females on emerging insects for food. Further, nest losses (e.g., due to predation) force many females to re-nest 1 or more times during the breeding season, thereby increasing the amount or time that females require high-protein invertebrate diets to meet the nutrient demands. And finally, decreased insect and invertebrate populations may force overland movement of females and their broods in search of adequate food, thereby increasing susceptibility to predation.

#### Possible Synergistic Effects with Other Pesticides



Interaction of ethyl parathion with other pesticides, e.g. imidazoles and dicarboximides, can increase acute toxicity. Based on studies with Japanese quail, hybrid red-legged partridge and pigeon, the acute toxicity of parathion, malathion, and dimethoate were enhanced following pretreatment with the fungicide prochloraz (Ronis et al. 1995). Also, Ronis et al. (1995)<sup>9</sup> showed a decrease in butyryl cholinesterase when ethyl parathion was combined with propiconazole and vinclozolin. Because ethyl parathion is often used with other pesticides during the same growing season, RQs derived from toxicity studies for ethyl parathion alone may underestimate the risk to birds.

In addition to fungicides, the use of ethyl parathion in combination with other organophosphates may result in potentiation of effect. Specifically, when 2 or more organophosphates are absorbed simultaneously, enzymes critical to the degradation of 1 may be inhibited by the other (Morgan, 1989). This is particularly important because 1 ethyl parathion product is formulated with methyl parathion. Also, Gordon et al. (1978) found that treatment of laboratory birds with a carbamate and then an organophosphate resulted in a 3- to 8-fold increase in toxicity of the organophosphate. Therefore, ethyl parathion's very high toxicity can be even greater under actual use conditions.

#### Indirect Evidence of Ecological Effects Based on a Comparison with Methyl Parathion

Ethyl parathion and methyl parathion share a common mode of action. Both are cholinesterase inhibitors which are converted to oxons in the environment and in animal livers. Büchel (1983) reported that methyl parathion is practically equivalent to ethyl parathion in activity. Moreover, ethyl parathion (LD 50 = 1 mg/kg) is more acutely toxic to birds than methyl parathion (LD50 = 10 mg/kg). Although there are less sublethal effects data available for ethyl parathion, the data for methyl parathion suggest strongly that sublethal effects to birds are likely to occur from ethyl parathion use.

Among the effects documented for methyl parathion and likely to occur as a result of ethyl parathion use are the following:

**Maternal behavioral changes** - Brood abandonment by hens (wood duck and teal) and nesting hen mortality occurred in methyl parathion treated fields. Also, brain cholinesterase levels were significantly depressed in 2 of 3 nesting hens. (Brewer et al. 1988; MRID 44371604)

**Covey integrity** - Buerger et al. (1991; MRID 44371606) indicated that bobwhite quail covey integrity may be at risk in a methyl parathion field study where treated birds had higher mortality due to predation than untreated birds. Individual birds not protected in a covey have much lower survival rates than birds retained in coveys.

**Anorexia and dietary discrimination** - Grue (1982; MRID 00311892) reported that behavioral and physiological responses of common grackles to dietary concentrations of dicotophos, fenitrothion, fenthion, and methyl parathion suggest mortality was largely due to pesticide-induced anorexia. Also, Mineau (1991) reported that two-week old northern bobwhite quail did not discriminate between untreated food and dietary containing 45 or 90 ppm methyl parathion, and initially (0-24 hours post

dose) chose treated over untreated food. This suggests that birds may not be able to select against ethyl parathion-contaminated food.

### **Effects on Reptiles**

Birds are OPP's surrogate for reptiles. Because of their inability to fly out of a contaminated area it is highly likely they are directly sprayed more often than birds and eat contaminated food more often. Therefore, EFED believes reptiles are at a similar or greater risk of adverse effects from ethyl parathion exposure than birds.

### **ii. Mammalian Assessment**

Estimating the potential for adverse effects to wild mammals is based upon EEB's draft 1995 SOP of mammalian risk assessments and methods used by Fletcher *et al.* (1994). The RQ is determined by dividing the EEC by the estimated LD50 dose per day. Risk quotients are calculated for 3 separate weight classes of mammals (15, 35, and 1000 g), each presumed to consume 4 different kinds of food (grass, forage, insects, and seeds). Each mammalian weight class is assumed to consume a different percent of their bodyweight daily, as seen in the tables below. The smallest mammals will eat a greater daily amount of food in proportion to their body weight, which is reflected in higher RQs than for larger mammals.

Parathion is "very highly toxic" (Brooks et al, 1973) to mammals on an acute basis (LD50=2.52 for female rats). The acute mammalian RQs for ethyl parathion are tabulated below.

Mammalian (Herbivore/Insectivore) Acute Risk Quotients for Single Application of Ethyl Parathion Based on a Rat LD50 of 2.52 mg/kg .

Site/ Application Method/ Rate in lbs ai/A	Body Weight (g)	% Body Weight Consumed	Rat LD50 (mg/kg)	EEC (ppm) Short Grass	EEC (ppm) Forage & Small Insects	EEC (ppm) Large Insects	Acute RQ <sup>1</sup> Short Grass	Acute RQ Forage & Small Insects	Acute RQ Large Insects
Alfalfa Canola									
0.5	15	95	2.52	120	67.5	7.5	45.2	25.4	2.8
0.5	35	66	2.52	120	67.5	7.5	31.4	17.7	2.0
0.5	1000	15	2.52	120	67.5	7.5	7.1	4.0	0.4
Barley Corn Soybeans Wheat									
0.75	15	95	2.52	180	101.25	11.25	67.9	38.2	4.2
0.75	35	66	2.52	180	101.25	11.25	47.1	26.5	2.9
0.75	1000	15	2.52	180	101.25	11.25	10.7	6.0	0.7
Cotton Sorghum Sunflower									
1	15	95	2.52	240	135	15	90.5	50.9	5.7
1	35	66	2.52	240	135	15	62.9	35.4	3.9
1	1000	15	2.52	240	135	15	14.3	8.0	0.9

$$^1 \text{ RQ} = \frac{\text{EEC (ppm)}}{\text{LD50 (mg/kg) / \% Body Weight Consumed}}$$

For single application scenario with herbivores/insectivores only the lowest application rate and those animals consuming only 15% of their bodyweight daily exceeds the endangered species and restricted use LOCs. All other scenarios also exceed the high risk LOCs.

Mammalian (Granivore) Acute Risk Quotients for Single Application of Ethyl Parathion Based on a Rat LD50 of 2.52 mg/kg.

Site/ Application Method/Rate in lbs ai/A	Body Weight (g)	% Body Weight Consumed	Rat LD50 (mg/kg)	EEC (ppm) Seeds	Acute RQ <sup>1</sup> Seeds
Alfalfa Canola					
0.5	15	21	2.52	7.5	0.6
0.5	35	15	2.52	7.5	0.4
0.5	1000	3	2.52	7.5	0.1
Barley Corn Soybean					
0.75	15	21	2.52	11.25	0.9
0.75	35	15	2.52	11.25	0.7
0.75	1000	3	2.52	11.25	0.1
Cotton Sorghum Sunflower					
1	15	21	2.52	15	1.3
1	35	15	2.52	15	0.9
1	1000	3	2.52	15	0.2

<sup>2</sup> RQ =  $\frac{\text{EEC (ppm)} * \% \text{ Body Weight Consumed}}{\text{LD50 (mg/kg)}}$

For granivores and the 0.75 and 1.0 lbs a.i./A rates, RQs exceed all LOCs except for the animals eating less than 3% of their body weights. The 3% consumers only exceed the endangered species LOC. For 0.5 lbs a.i./A application rate the 21% consumer exceed all LOCs, 15% consumer exceeds both endangered species and restricted use, and 3% consumer does not exceed any of the LOCs.

Mammalian Chronic Risk Quotients for Single Applications of Ethyl Parathion Based on a Rat NOEC of 2.52 ppm in a Feeding Study and Rat NOEC of 10 ppm in a Reproduction Study.

Site ( # of Apps. /seasons) (Interval between App)	lbs a.i./A	Food Items	Maxi- mum EEC (ppm)	Reproductive Study NOEC (ppm)	Reproductive RQ (EEC/ NOEC)	Reproductive LOC <sup>3</sup> Exceedance
Alfalfa Canola (2)(7)	0.5	Short grass	120	10	12.0	All
		Tall grass	55	10	5.5	All
		Broadleaf plants/ Insects	77	10	7.7	All
		Seeds	8	10	0.8	All
Soybean (2)(7) Barley Wheat (6)(7) Corn (6)(5)	0.75	Short grass	180	10	18.0	All
		Tall grass	83	10	8.3	All
		Broadleaf plants/ Insects	101	10	10.1	All
		Seeds	9	10	0.9	All
Sunflower (3)(5) Cotton Sorghum (6)(7)	1	Short grass	240	10	24.0	All
		Tall	110	10	11.0	All
		Broadleaf plants/ Insects	135	10	13.5	All
		Seeds	15	10	1.5	All

<sup>1</sup> Each percent body weight consumed is representative of a different size animal. 21%, 15%, and 3% are for 15 g, 35 g, and 1000 g animals respectively.

<sup>2</sup>  $RQ = \frac{EEC (ppm) * \% \text{ Body Weight Consumed}}{LD50 (mg/kg)}$

<sup>3</sup> LOC = 1

### Acute risk

Acute risk from the ingestion of ethyl parathion is expected, based on the RQs above. These RQs may underestimate the risk, based on information from human incidents. In one incident, children aged 5 to 6 have were killed from eating 2 mg of parathion, which was a dose of about 0.1 mg/kg. Another fatal human poisoning resulted from an ingested rate of 1.7 mg/kg (USEPA,1991). The acute RQs above were calculated with the rat LD50 of 2.52 mg/kg. If the 0.1 mg/kg dose were used instead, the resulting 0.5 lb ai/A RQ would rise from 0.09 to 2.25 for the largest mammals tested, resulting in exceedances of all acute LOCs.

Wildlife incident reports confirm that ethyl parathion use has caused mortality to mammals of different size (e.g., humans, horses, rabbits, squirrels and domestic pigs). Incident reports include 1.) 2 horses died from eating ethyl parathion contaminated hay, 2.) pregnant sows died after application of ethyl parathion to sunflowers, 3.) 2 rabbits died from ethyl parathion use on cotton, and 4.) Several squirrels died from ethyl parathion use on a cotton field. In addition to wildlife incident reports, there are numerous human incident reports of sublethal effects to death from ethyl parathion (See attached Notice of Intent to Cancel). Incidents may understate the magnitude of the impact on small mammals. Small mammal carcasses are usually not visible and thus go unnoticed.

The risk of ethyl parathion exposure to mammals is likely to be increased by inhalation and dermal exposure, and also oral exposure through grooming. The only available inhalation study for wild mammals is a supplementary rat study showing that ethyl parathion LC50 is above 1.3 mg/L. However, an extensive database exists showing lethal and sublethal human poisonings through ethyl parathion exposure prior to the 1991 agreement between Cheminova and EPA. Mitigation put into effect by that agreement, such as reentry restrictions and protective clothing requirements, were in response to exposure by ingestion, dermal contact and inhalation of ethyl parathion. These measures have been successful in curtailing human poisonings in the field. However, exposure to wild mammals is not affected by these mitigation measures, and extensive mammalian exposure is expected with a high degree of certainty.

### Chronic risk

Mammals that survive exposure to ethyl parathion are likely to suffer adverse reproductive effects. As shown above, the reproductive mammalian RQs exceed LOCs after a single application of ethyl parathion. Multiple applications are expected to increase the risk, especially for the crops which allow 6 applications in a growing season (barley, corn, cotton, sorghum, and wheat). As demonstrated by the feeding and reproduction studies, the dose required to cause effects is expected to decrease with increasing exposure periods. In addition, each additional application is another opportunity for intoxication at a critical point in the development of an unborn animal.

### iii. Bees and Other Pollinators Assessment

As stated above, toxicity testing indicates that ethyl parathion is very highly toxic to bees. Seven reported bee kills associated with application of ethyl parathion to sunflowers are summarized in Table 3 of the Appendix.

In addition, the American Beekeeping Federation, Inc. did a survey of its members to determine the extent of damage to bee colonies due to pesticide exposure. This survey was compiled through June 16, 1997. Sixty beekeepers, operating 127,950 colonies in 22 states, reported that bee losses from pesticides are a significant issue in their operations. The survey listed the pesticides in order according to number of bee kill responses as follows: Ferritin, PennCap-M, Sevin, and ethyl parathion. This indicates further that current uses of ethyl parathion poses acute risk to bees.

This assessment is important because pollinators (bees, wasps, bumble bees, etc) fill an important ecological niche. They transfer pollen between plants, helping to ensure fruit and vegetable growth and seed viability. Pollinators can be very specialized. For example, the alkali bee is especially apt at opening the alfalfa flower and extracting pollen. Therefore, loss of specific pollinators can change ecological relationships that can reduce the success of a given plant and make unintended changes in the flora. Changes in the flora may also affect the animal population which relies on the plant for cover, nesting, feeding, etc.

### b. Exposure and Risk to Nontarget Freshwater Aquatic Animals

EFED calculates EECs using GENEEC and PRZM./EXAMS (see discussion above). Acute aquatic risk assessments are performed using peak EEC values for single and multiple applications. Chronic aquatic risk assessments are performed using the 21-day EECs for invertebrates and 56-day EECs for fish.

## i. Freshwater Fish

Acute and chronic RQs tabulated below are based on a bluegill sunfish LC50 of 18 ppb and fish early-life stage study NOEC of 0.17 ppb.

## Risk Quotients for Freshwater Fish

Site/ Rate in lbs ai/A (No. of Apps.) (Interval)	LC50 (ppb)	NOEC (ppb)	EEC Initial/ Peak (ppb)	EEC 56 or 60 Days Ave. (ppb)	Acute RQ (EEC/ LC50)	Chronic RQ (EEC/ NOEC)
<b>GENEEC</b>						
Canola 0.5 (2)(7)	18	0.17	30.2	4.2	1.7	24.8
Sunflower 1.0 (3)(5)	18	0.17	132.7	12.5	7.4	73.3
Barley Wheat 0.75 (6)(5)	18	0.17	124.5	17.3	6.9	101.8
<b>PRZM/ EXAMS</b>						
Alfalfa 0.5(8)(7)	18	0.17	4.7	1.6	0.3	9.4
0.5(2)(7)	18	0.17	1.0	0.40	0.1	2.4
Corn 0.75 (6)(5)	18	0.17	39.8	13.4	2.2	78.8
Cotton 1.0(6)(7)	18	0.17	54.7	20.2	3.0	118.8
Sorghum 1.0(6)(7)	18	0.17	60.9	22.3	3.4	131.1
Soybean 0.75 (2)(7)	18	0.17	13.9	4.7	0.8	27.6

All LOCs for freshwater fish both acute and chronic risk have been exceeded except for an alfalfa application of 0.5 lbs a.i./A RQ. However, acute alfalfa RQ exceeds both restricted use and endangered species LOCs. As noted above, the number of applications (8) simulated for alfalfa by PRZM-EXAMS is consistent with current label language. Cheminova has proposed a tolerance for



alfalfa that would allow only 2 applications of 0.5 lb ai/A per year. The acute RQ resulting from only 2 applications exceeds the endangered species LOC.

Sublethal exposure to organophosphates (OP) can be expected to change behavior. OP exposure can cause hyperactivity, muscular spasms, and tetany in fish. Henry (1984)(MRID No.:44371607) observed these effects in bluegills after exposure to methyl parathion. He suggested that survival and reproduction of individuals in a natural population could be affected “if associated courtship, territoriality, aggression, feeding and comfort movements are disrupted”. Since ethyl parathion has the same mode of action, and is much more toxic to bluegills than methyl parathion (LC50 of 18 ppb versus methyl parathion’s bluegill LC50 of 1000 ppm), ethyl parathion exposure may cause such population effects, as well. However, as opposed to more dramatic effects such as fish kills, this kind of population decline would likely go unnoticed unless the location was under scientific observation.

As mentioned under the avian discussion, tadpoles concentrate parathion and are relatively tolerant to ethyl parathion exposure. Based on this, consumption of tadpoles exposed to ethyl parathion may be toxic to aquatic predators. (Stansell, 1993)

## ii. Freshwater Invertebrates

The freshwater invertebrate acute and chronic RQs tabulated below are based on a crayfish (*Orconectes nais*) EC50 of 0.04 ppb and a water flea NOEC of 0.002 ppb.

### Risk Quotients for Freshwater Invertebrates

Site/ Rate in lbs ai/A (No. of Apps.) (Interval)	LC50 (ppb)	NOEC (ppb)	EEC Initial /Peak (ppb)	EEC 21-Day Average (ppb)	Acute RQ (EEC/LC50)	Chronic RQ (EEC/NOEC)
<b>GENEEC</b>						
Canola 0.5 (2)(7)	0.04	0.002	30.2	10.6	755.5	5310.0
Sunflower 1.0 (3)(5)	0.04	0.002	132.7	31.4	3316.5	15700.0
Barley Wheat 0.75 (6)(5)	0.04	0.002	124.5	43.6	3111.8	21795.0
<b>PRZM/ EXAMS</b>						
Alfalfa 0.5 (8)(7)	0.04	0.002	4.6	2.9	115.0	1450.0
0.5(2)(7)	0.04	0.002	1.0	0.7	25.0	350.0
Corn 0.75 (6)(5)	0.04	0.002	39.8	25.7	995.0	12850.0
Cotton 1.0 (6)(7)	0.04	0.002	54.7	33.1	1367.5	16550.0
Sorghum 1.0 (6)(7)	0.04	0.002	60.9	37.4	1522.5	18700.0
Soybean 0.75 (2)(7)	0.04	0.002	13.9	8.6	347.5	4300.0

All freshwater invertebrates acute and chronic RQs exceed all LOCs. Ethyl parathion is very highly toxic to freshwater invertebrates.

These data suggest that the use of ethyl parathion can lead to acute risk to estuarine invertebrates which could lead to significant effects to higher aquatic organisms which feed on the invertebrates. Crossland (1984;MRID No.: 44371714) suggested exposure of mayflies and daphnids in a methyl

parathion-treated pond led indirectly to a fish kill. The elimination of these invertebrate predators led to an algae bloom which eventually depleted dissolved oxygen in the pond, killing the fish.

Given the common mode of action between methyl parathion and ethyl parathion, and the fact that ethyl parathion is toxic to freshwater invertebrates at lower concentrations, it is likely that ethyl parathion could also cause such ecological effects.

### c. Estuarine and Marine Animals

The acute and chronic RQs tabulated below are based on a spot LC50 of 18 ppb and sheepshead minnow NOEC of 0.19 ppb.

#### Risk Quotients for Estuarine/Marine Fish

Site/ Application Method	Rate in lbs ai/A (No. of Apps.)(Interval between Apps.	LC50 (ppb)	NOEC (ppb)	EEC Initial/ Peak (ppb)	EEC 56-60 Day Ave (ppb)	Initial RQ (EEC/ LC50)	56-day RQ (EEC/ NOEC)
GENEEC							
Canola	0.5(2)(7)	18	0.19	15.1	4.2	0.8	22.2
Sunflower	1.0(3)(5)	18	0.19	89.4	12.5	5.0	65.6
Barley Wheat	0.75(6)(7)	18	0.19	124.5	17.3	6.9	91.1
PRZM/ EXAMS							
Alfalfa	0.5(2)(7)	18	0.19	4.7	1.6	0.3	8.4
		18	0.19	1.0	0.37	0.1	1.9
Corn	0.75(6)(5)	18	0.19	39.8	13.4	2.2	70.5
Cotton	1.0(6)(7)	18	0.19	54.7	20.2	3.0	106.3
Sorghum	1.0(6)(7)	18	0.19	60.9	22.3	3.4	117.4
Soybean	0.75(2)(7)	18	0.19	13.9	4.7	0.8	24.7

All estuarine and marine fish acute and chronic RQs exceed all LOCs except the PRZM/EXAMS value for alfalfa. The alfalfa PRZM/EXAMS scenario again exceeds both restricted use and endangered species LOCs. With only 2 applications the RQ exceeds only the endangered species LOC.

Risk Quotients for Estuarine/Marine Aquatic Invertebrates Based on a Mysid LC50 of 0.107 ppb and a Mysid NOEC of 0.0031 ppb.

Site/ Application Method	Rate in lbs ai/A (No. of Apps.)	EEC Initial/ Peak (ppb)	EEC 21-Day Average	Initial RQ (EEC/LC50)	21-Day RQ (EEC/NOEC)
GENEEC					
Canola	0.5(2)(7)	15.1	10.6	141.2	3425.8
Sunflower	1.0(3)(5)	89.4	31.4	835.1	10129.0
Barley Wheat	0.75(6)(7)	124.5	43.6	1163.3	14061.3
PRZM/EXAMS					
Alfalfa	0.5(8)(7)	4.7	2.9	44.0	935.5
Alfalfa	0.5(2)(7)	1.0	0.65	9.3	209.7
Corn	0.75(6)(5)	39.8	25.7	372.0	8920.3
Cotton	1.0(6)(7)	54.7	33.1	511.2	10677.4
Sorghum	1.0(6)(7)	60.9	37.4	569.2	12064.5
Soybean	0.75(2)(7)	13.9	8.6	129.9	2774.2

All estuarine and marine aquatic invertebrates acute and chronic RQs exceed all LOCs. Invertebrate PRZM/EXAMS acute RQs range from 36 to 339. Invertebrate PRZM/EXAMS chronic RQs range from 9.7 to 588. Acute and chronic risks to estuarine/marine invertebrates are high.

#### d. Exposure and Risk to Nontarget Plants

##### i. Terrestrial Plants

Currently, terrestrial plant testing is not required for pesticides other than herbicides except on a case-by-case basis (e.g. labeling bears phytoxicity warnings incident data or literature that demonstrate phytotoxicity). Hartley and Hamish (1987) indicates that ethyl parathion is “non-phytotoxic, except to some ornamentals, cucurbits, sorghum, and some varieties of apple, pear, and tomato.” Due to the demonstrated phytotoxicity to plants, Tier I terrestrial plants phytotoxicity tests are required (122-1, seedling emergence and vegetative vigor) in which the maximum registered label dosage is used. These studies will be useful in determining the risk to endangered plants.

##### ii. Aquatic Plants

Aquatic plants testing is required for the any pesticide with a phytotoxicity warning on the on product, incident reports of phytotoxic, or other sources (such as published literature) of reported phytotoxicity. These studies are important to show if the aquatic plant species are affected. These studies will be useful in determining the risk to endangered plants.

The following species should be tested at Tier I: *Kirchneria subcapitata*, and *Lemna gibba*. Aquatic plant testing is required for parathion because of demonstrated phytotoxicity to plants.

#### 10. ENDANGERED SPECIES

Endangered species LOCs for ethyl parathion are exceeded for birds, mammals, fish and invertebrates.

The Agency has developed a program (the “Endangered Species Protection Program”) to identify pesticides whose use may cause adverse impacts on endangered and threatened species, and to implement mitigation measures that will eliminate the adverse impacts. At present, the program is being implemented on an interim basis as described in a Federal Register notice (54 FR 27984-28008, July 3, 1989), and is providing information to pesticide users to help them protect these species on a voluntary basis. As currently planned, the final program will call for label modifications referring to required limitations on pesticide uses, typically as depicted in county-specific bulletins or by other site-specific mechanisms as specified by state partners. A final program, which may be altered from the interim program, will be described in a future Federal Register notice. The Agency is not imposing label modifications at this time through the RED. Rather, any requirements for product use modifications will occur in the future under the Endangered Species Protection Program. Currently available county specific information, maps and a downloadable version of the Endangered Species data base can be found on the Internet at the Agency's web site, <http://www.epa.gov/ESPP>.

## 11. RISK CHARACTERIZATION

### a. Introduction

EFED concludes with a great deal of certainty that the use of ethyl parathion poses a high risk to nontarget organisms in terrestrial and aquatic environments. This assessment is based on laboratory toxicity data linked to terrestrial and aquatic exposure models, incident data, and comparative analysis with structurally similar compounds with common modes of action. The toxicological and exposure data suggest strongly that acute and chronic effects on birds and mammals, acute effects on bees, and acute and chronic effects on aquatic invertebrate organisms are likely to occur as a result of ethyl parathion applications. The impact of ethyl parathion use on freshwater fish is less certain because the risk analysis indicates slight exceedance of the levels of concern. Monitoring data include detections of ethyl parathion residues in ground and surface water, but suggest that the risk of drinking water exposure is less than that predicted by simulation models.

The incident data compiled for ethyl parathion confirm adverse effects to both humans and wildlife (see attachments). There are extensive incident data linking ethyl parathion to accidental and intentional poisoning of humans as reported in OPP's Notice of Intent to Cancel. These poisonings include sublethal effects, and in some cases, mortality. Wildlife incident data link bird and mammal mortality to ethyl parathion use. These exposures have been associated with labeled uses, accidental exposures, and intentional misuses of ethyl parathion.

#### Relevance of Data from Methyl Parathion

Considered by itself, laboratory, field, and incident data indicate that ethyl parathion use will result in effects in non-target organisms. This conclusion is bolstered by consideration of toxicity data for methyl parathion, a structurally similar organophosphate pesticide. Although methyl parathion is less toxic than ethyl parathion, both compounds are cholinesterase inhibitors (Büchel 1983). The difference in toxicity is related to the compounds' polarity; the ethyl moiety of ethyl parathion creates a greater polarity which creates a higher binding affinity for phosphorylation of the acetylcholinesterase enzyme at the nerve synapses. Because the Food Quality Protection Act (FQPA) requires that aggregate exposure from pesticides with a common mode of action be incorporated into human risk assessment, a similar type of assessment is relevant to understanding ecological risk from the use of ethyl parathion.

Environmental fate data suggest that, in addition to being more toxic, ethyl parathion is more persistent than methyl parathion. Although both compounds have similar degradation pathways (*e.g.*, microbial-mediated hydrolysis and oxidative desulfonation) in terrestrial and aquatic environments, methyl parathion appears to degrade much faster than ethyl parathion. Both compounds, however, exhibit a moderate binding affinity to soil organic matter.

### Data Gaps

Through the risk assessment and characterization processes, several major data gaps have been identified in understanding the exposure profile of ethyl parathion and its impact on non-target organisms. The outstanding environmental fate data requirements for ethyl parathion include Anaerobic Aquatic Metabolism (162-3), Aerobic Aquatic Metabolism (162-4), Batch Equilibrium Soil Column Leaching (163-1), Terrestrial Field Dissipation (164-1), Aquatic Field Dissipation (164-2), and Accumulation in Fish (165-4). A common data gap in these studies is associated with the analytical methods used for identification and quantification of ethyl parathion degradation products and the lack of confirmatory storage stability data. Since these problems can not be resolved through submission of additional data, new studies will be needed to confirm supplemental data used in the exposure assessment. In addition, a major data gap in the environmental fate assessment is the lack of fate and transport data for ethyl paraoxon.

Although foliar dissipation studies are not routinely required, a complete environmental assessment for ethyl parathion and its degradates requires an understanding of the routes and rates of dissipation from foliage. This information is needed because ethyl parathion is applied to foliage. The outstanding ecotoxicology data requirements are plant toxicity tests: 122-1(a) Seed Germination/Seedling Emergence, 122-1(b) Vegetative Vigor, and 122-2 Aquatic Plant Growth. These data are needed to adequately address toxicity issues in aquatic and terrestrial ecosystems.

## b. Drinking Water

### i. Surface Water

Direct drinking-water data for ethyl parathion are not readily available, and it is not likely that much of such data has been collected. The Office of Water has not established a Maximum Contaminant Level (MCL) for ethyl parathion and its degradate ethyl paraoxon, and they are not included on the Unregulated Contaminant Monitoring List. Therefore, public drinking water supply systems are not required to analyze for ethyl parathion. Although ethyl parathion was sporadically detected in the USGS National Water Quality Assessments (NAWQA) monitoring program, the quality of the data are suspect because of low analytical recoveries. Additionally, there are no monitoring data to assess the presence of ethyl paraoxon in drinking or surface waters. Consequently, EFED relied predominately on simulation models for predicting concentrations of ethyl parathion in drinking water and aquatic exposure assessments. The lack of environmental fate and transport data for ethyl paraoxon prevented the use of models for prediction of its concentration in aquatic environments and drinking water.

The PRZM-EXAMS screening models predict that surface water concentrations of ethyl parathion are not likely to exceed 60.9 ppb for peak (acute) and 5.4 ppb for annual time weighted mean (chronic). Although these screening estimates are higher than the concentrations seen in monitoring studies, the observed difference can be attributed in part to the conservative nature of the models themselves. As detailed in the drinking water assessment above, the assumptions are intentionally conservative to ensure the maximum protection of human health.

There is high uncertainty in the drinking water and aquatic exposure assessments for ethyl parathion. Factors contributing to the uncertainty are attributed to: 1.) inherent conservativeness of the modeling scenario used for the drinking water assessment; 2.) the inability to evaluate fate and transport of ethyl paraoxon, a toxicological important degradate, and 3.) the lack of monitoring data for ethyl parathion and ethyl paraoxon in drinking water.



## ii. Acute Drinking Water Exposure

Data from targeted monitoring studies might provide a better estimate of possible acute drinking water concentrations than the models. However, this kind of monitoring data is not available for ethyl parathion, with the possible exception of an 1985 urban runoff study performed near an area of heavy agricultural use of ethyl parathion. This study (Oltmann, et al., 1985, cited in Larson, et al., 1997) reported a maximum detection of 2.5 ppb ethyl parathion. In addition, fifty-seven tile drain and other agricultural discharge samples in the San Joaquin and Tulare basins taken by the California Department of Water Resources between 1966 and 1992 yielded few detections, with a maximum concentration of 0.9 ppb. It must be noted that these studies took place before current mitigation practices went into effect.

Since targeted monitoring studies are not available in connection with ethyl parathion uses, surface-water concentrations simulated with PRZM-EXAMS for drinking water assessments should be considered conservative, but should not be arbitrarily reduced. The conservativeness of the EECs should be considered when developing mitigation to protect human health, non-target organisms, and water resources. Potential mitigation measures are detailed at the end of the risk characterization section.

## iii. Chronic Drinking Water Exposure

Non-targeted surface-water survey studies performed over 30 years have not shown concentrations of ethyl parathion at chronic levels predicted in modeling assessments. While maximum measured concentrations reported from available studies were below the range of chronic EECs predicted by PRZM-EXAMS (0.12 to 5.4 ppb), these represent isolated detections in surface water, and not chronic contamination. For instance, the results of the more recent studies in the NAWQA program resulted in maximum concentrations of 0.14 ppb in agricultural streams, and 0.014 ppb in urban streams. However, of the 1000 agricultural stream samples reported, only two samples (0.20%) had detections of ethyl parathion, and among these the 95<sup>th</sup> percentile concentration was below the detection limit. Since the recent studies are not specifically targeted to ethyl parathion use, it is difficult to interpret the monitoring data in accordance with current ethyl parathion uses. Additionally, the analytical recovery for ethyl parathion in the NAWQA samples was low (58% recovery) which further limits quantitative interpretation of the data.

Although available monitoring data do not allow a definitive assessment, they reinforce the notion that the PRZM-EXAMS EECs should be considered conservative when considering further mitigation based on surface-water concerns.

## iv. Ground Water

The SCI-GROW screening model predicts that ethyl parathion concentrations in shallow ground water are not likely to exceed 1.21 ppb. Data collected from a variety of sources did not identify any known instance in which a ground-water concentration was higher than 1.21 ppb, with the single

exception of a suspect detection in Georgia of 99 ppb reported in the Pesticides in Ground Water Database. Therefore, EFED suggests that 1.21 ppb is a reasonable conservative estimate of possible acute concentrations of ethyl parathion in drinking water derived from shallow ground water.

Since ethyl parathion has been rarely detected in ground-water in all studies evaluated, the SCI-GROW concentration of 1.21 ppb is likely an upper bound estimate for chronic risk assessments. For instance, ethyl parathion was not found in the 1130 samples taken between 1991 and 1995 in the USGS NAWQA study. Since the recent studies are not specifically targeted to ethyl parathion use, it is difficult to interpret the monitoring data in accordance with current ethyl parathion uses. Additionally, the analytical recovery for ethyl parathion was low (58% recovery) which further limits quantitative interpretation of the data.

EFED does not have a tool for estimating second-tier ground water concentrations for dietary risk assessments. However, environmental fate data suggest that ethyl parathion has relatively high soil:water partitioning coefficients ( $K_d=9.1$  to  $25.3$  ml/g) in fine textured soils. It should be noted that ethyl parathion has a low soil:water partitioning coefficient ( $K_d < 5$  ml/g) in sand. Since the SCI-GROW model is based on data from prospective ground water studies conducted on coarse textured soils, it is reasonable to believe the SCI-GROW estimate is an upper bound estimate of long-term concentrations of ethyl parathion in ground water.

### c. Ecological Effects

#### i. Avian Risk Characterization

EFED concludes the available fate and effects data suggest that ethyl parathion poses an acute and chronic risk to birds. This is founded on (1) a consistent toxicological database showing high avian toxicity to ethyl parathion, (2) an extensive incident database showing adverse effects to birds linked to ethyl parathion use, (3) the potential for formation of a highly toxic degradation product (ethyl paraoxon), and (4) the widespread use of the compound on crops that are attractive to wildlife.

Besides acute mortality, ethyl parathion is likely to result in sublethal effects on birds such as:

- ! reproduction effects,
- ! endocrine disruptive properties,
- ! greater sensitivity to environmental stress.

Although other sublethal effects have not been documented for ethyl parathion, registrant and open literature data for methyl parathion, a close chemical analog with a mode of toxic action in common with ethyl parathion, suggest other potential acute sublethal effects may be possible including:

- ! reproductive effects from acute exposure,
- ! changes in maternal care and viability of young birds,
- ! increased susceptibility to predation.

As stipulated by FQPA, the risk posed by different pesticides with the same mode of action must be considered together. A similar assessment is needed for the avian risk assessment because the risk associated with the use of ethyl parathion may be compounded by other organophosphates, which share a common mode of action (cholinesterase inhibition). The EC combination of ethyl parathion with methyl parathion is the most obvious example. This product is likely to present more risk than if ethyl parathion is used alone. Additionally, there are potential synergistic toxicological effects from the interaction of ethyl parathion with the fungicide, prochloraz (Ronis et al 1995). Although this assessment does not quantitatively address the interactive effects of ethyl parathion with other pesticides, there is a potential for multiple avian exposures to numerous pesticides, which may result in enhanced toxicological effects.

Extensive incident data confirm that 1 application of ethyl parathion can cause avian mortality in the field. It has been suggested by the registrant that enhanced stewardship with respect to the use of ethyl parathion has contributed to observed reductions in incidents after 1991. EFED has insufficient information to determine the validity of this assertion. However, EFED is aware that actions to protect farm workers in treated fields (e.g., extended post application entry intervals) would reduce the opportunity to actually observe avian mortality or morbidity in treated fields.

Toxicity tests show that a short exposure to ethyl parathion can cause adverse reproduction effects in surviving birds. The Agency believes that the available data are compelling for a conclusion that ethyl parathion use poses acute and reproduction risk to birds and other terrestrial organisms. Although residue dissipation was not considered in the RQ analyses, any risk reducing effect through dissipation would be offset due to the following:

1. Laboratory diets contain less water and have greater caloric content than diets in the wild. This would cause laboratory animals to consume less food and thus ingest less pesticide in the laboratory compared to in the field.
2. Animals in the laboratory have a lower metabolic rate than those in the wild due to being inactive and being in a temperature-controlled environment. This would cause laboratory animals to consume less food and thus ingest less pesticide in the laboratory compared to in the field.
3. Wildlife in the laboratory studies are exposed only through ingestion of the chemical in the diet, whereas wildlife would also be exposed through other routes (e.g. dermal absorption, inhalation, drinking, and preening).
4. Unlike animals in the laboratory wildlife are exposed to stressors other than chemical toxicity (e.g. heat and cold, disease, parasites, malnutrition, and predation pressure). These additional stressors may make wildlife less tolerant of chemical stressors.

Multiple applications of ethyl parathion can theoretically cause some accumulation of ethyl parathion on foliage even when a foliar dissipation rate of 2.1 days is considered. This will increase the likelihood of longer-term exposure. Based on the average foliar dissipation half-life, the label maximum of 6 applications at 1.0 lb ai /A (weekly interval) results in avian food item residues

exceeding reproduction levels of concern for more than 7 weeks. These data suggest the magnitude and duration of ethyl parathion concentrations on foliage are sufficient to cause subacute and chronic effects when considering the mean foliar dissipation rate. More residue accumulation and prolonged effects are expected for longer foliar dissipation half-lives (*e.g.*, upper 90<sup>th</sup> percentile). Because this avian exposure assessment is based solely on ethyl parathion and not cumulative ethyl parathion residues (*e.g.*, ethyl parathion and ethyl paraoxon), it is likely the actual duration and magnitude of exposure to toxic ethyl parathion residues is underestimated.

Beside dietary exposure, avian species are exposed to ethyl parathion through inhalation and dermal adsorption (Stavola, 1987). Although this risk assessment does not quantitatively account for inhalation exposures, it is possible that volatilized ethyl parathion as well as ethyl parathion on respirable soil particles contributes to the overall body burden in wildlife exposed to ethyl parathion. Although the physicochemical data for ethyl parathion suggests it is not highly volatile (Henry's Constant  $6.7 \times 10^{-7}$  atm-m<sup>3</sup>/mole), ethyl parathion has been detected in air monitoring studies at concentrations of 1.1 to 239 ng/m<sup>3</sup> (Majewski and Capel, 1995). Incident data indicate that sublethal effects (*e.g.*, vomiting, nausea, headaches, dizziness, throat irritation, and stomach) in humans have been linked to inhalation exposure. It is likely that smaller unprotected animals, which have higher respiration rates than humans and will spend considerable time in treated fields in close proximity to treated soils and foliage, will be exposed to equivalent or greater levels ethyl parathion through the inhalation route. Another potential, but unquantifiable, route of exposure is associated with dermal adsorption of ethyl parathion and its degradate ethyl paraoxon. Ethyl parathion has been shown to be a potent toxicant via dermal adsorption. (The dermal acute LD<sub>50</sub> for house sparrow and quelea is 1.8 mg/kg-bw.) This level of toxicity suggests that low residue concentrations in the field can cause adverse effects. Additionally, the ethyl parathion degradate, ethyl paraoxon, is more toxic (40 to 50X) and more readily adsorbed than ethyl parathion (USEPA, 1991).

Incident data confirm that dermal and inhalation, in addition to dietary exposure, cause adverse effects to non-target organisms. Incidents with geese that graze on winter wheat treated with ethyl parathion are typical of oral or dietary exposure. However, 4 separate incidents involving domestic chickens, barn swallows, red tailed hawks, and purple martins were reported after initial exposure to a spray mist of ethyl parathion. In addition, there are incident data showing that secondary exposure of non-target organism via contaminated prey can cause adverse effects. Incidents of secondary exposure include 1.) mortality of kites feeding on contaminated insects, 2.) mortality of bald eagle and red tailed hawk feeding on contaminated prey, 3.) mortality of mallard ducklings feeding on tadpoles with bioconcentrated ethyl parathion (Hall et al. 1980), and 4.) mortality of American kestrels (*Falco sparverius*) feeding on crickets frogs with bioconcentrated ethyl parathion (Fleming et al., 1982).

The incidents show that ethyl parathion can be easily abused. There were several incidents of intentional and/or misuse bird poisonings. These incidents were associated with 1.) nonpest birds feeding on contaminated grain used to attract and kill pest birds and 2.) spraying of crops within wildlife refuges.

Finally, the environmental fate database for the toxic degradate of ethyl parathion, ethyl paraoxon, is incomplete to assess environmental concentrations in terrestrial environments. The exclusion of ethyl paraoxon from the terrestrial exposure assessment represents an *underestimation* of avian and

mammalian exposure to biologically active ethyl parathion residues. Registrant and open literature data indicate that ethyl paraoxon can form in air, soil, water and on foliage when treated with ethyl parathion. The presence of ethyl paraoxon in numerous environmental compartments provides a higher probability for multiple routes of avian exposure.

### Avian Geographical/Regional Considerations

Because ethyl parathion is used on several major crops (*e.g.*, wheat, cotton, and corn), there is an increased chance for avian exposure. The majority of ethyl parathion is used in the Great Plains region of the United States (North Dakota, South Dakota, Nebraska, Kansas, northeastern Colorado, Oklahoma, east central counties of New Mexico, and the Texas panhandle) and the coastal counties of Texas. There are 7 other states with pockets of high use including Arizona, Georgia, Alabama, Washington, Montana, Arizona, and Delaware. The highest use areas are associated with the Great Plains region (North Dakota, Kansas, and Oklahoma), and Georgia (Cheminova, 1992). These use areas are associated with the production of sunflowers, sorghum, corn, alfalfa, and cotton.

The widespread geographical extent of ethyl parathion use area suggests a high potential for adverse effects to diverse species of birds. Dunning (1984) indicates that there are 686 bird species in North America, which include both migratory and resident birds. Incident data confirm effects on a wide variety of avian species including rock doves, kites, owls, purple martins, barn swallows, pheasants, grackles, red tailed hawks, starlings, blue grosbeak, eastern bluebirds, Franklin gulls, cedar waxwings, blacknecked stilt, goldfinch, bandtail pigeons, turkeys, domestic chickens, laughing gull, bald eagle (threatened species) and prairie chickens.

Although ethyl parathion use on corn is predominately in Georgia, the label does not have a geographic use restriction. Therefore, ethyl parathion could be used anywhere in the corn production area of the United States. Corn is planted in 80 million acres in the United States. Because there are at least 200 species of birds found in and around corn fields (see Appendix, Table 4)(USEPA. 1996), the use of ethyl parathion in the corn production area of the United States could result in widespread exposure to both waterfowl and resident birds.

The use of ethyl parathion is expected to coincide with waterfowl breeding in the Central and Atlantic flyways. These flyways are major migratory routes for waterfowl between breeding and wintering grounds. The Central flyway is generally associated with Great Plain region of the United States, and the Atlantic flyway is associated with the eastern seaboard of the United States.

Ethyl parathion use is primarily concentrated along the Central flyway. Within this flyway, major breeding grounds for waterfowl are in the prairie-pothole region of North America, with the greatest concentration of breeding ducks per square mile found in the Dakotas (see Appendix, Figure 1). Grue, et al. (1988) reported that about 75% of cultivated land in North Dakota is in the prairie-pothole region where important crops include spring wheat, barley and sunflowers; ethyl parathion is used on each of these crops. The Texas' Gulf coast is a the primary destination for many Central flyway waterfowl. Ethyl parathion is used in Texas on the following crops: sunflowers, sorghum, corn, alfalfa, cotton, wheat, and barley.

In the Atlantic Flyway, ethyl parathion is predominately used in Delaware, Georgia, Florida, and Alabama (Cheminova, 1997). These states are wintering grounds for waterfowl. Because of the overlap of breeding and wintering grounds, Delaware has the highest concentration of breeding ducks in the Atlantic flyway.

## ii. Mammalian Risk Characterization

The risk quotients developed for this risk assessment suggest that some ethyl parathion use rates present an acute risk to small mammals. All herbivore RQs exceed the LOCs except for the large mammal (1000 g animal) at the lowest application rate (0.5 lb a.i./A). Chronic and reproduction RQs are exceeded for a single application of 0.5 lbs ai/A or greater. The herbivore and insectivore mammals acute RQs ranged from 0.45 to 90.48 for the lowest single application rate. The granivores acute RQs ranged from 0.09 to 1.25. The single-application LOCs for small (15 g) granivores are all exceeded at application rates of 0.5 lb ai/A. All LOCs for 35-gram granivores are exceeded for application rates of 0.75 lb ai/A or greater. The large mammal (1000 g animal) RQ exceeds only the endangered species LOC. These data suggest that lethal effects, and possibly sublethal effects, are possible from a single application of ethyl parathion. However, there is considerable uncertainty in extrapolating an acute dietary toxicity endpoint from available single oral dose data. Data from (McCann, 1981) suggest that such an approach with ethyl parathion may overestimate the acute dietary toxic potency by a factor of around 2X. If this held true for all small mammal species (there are no data to substantiate the nature of this extrapolation error across species) the resultant RQ calculated would be overestimated by a factor of approximately 2X. However, with the exception of granivore exposures, this factor would have little impact on the conclusions of the assessment. Furthermore, wildlife incident reports confirm that ethyl parathion use has caused mortality in mammals of different sizes (*e.g.*, humans, horses, rabbits, squirrels and domestic pigs). Incident reports include 1.) the death of 2 horses from eating ethyl parathion contaminated hay, 2.) the death of pregnant sows after application of ethyl parathion to sunflowers, 3.) the death of 2 rabbits from ethyl parathion use on cotton, and 4.) the death of several squirrels from ethyl parathion use on a cotton field. In addition to wildlife incident reports, there are numerous human incident reports effects linked to ethyl parathion exposure ranging from sublethal to lethal. (Please see attached Notice of Intent to Cancel.) Incident reports indicate that the lethal ethyl parathion dose rate for humans ranged from 0.1 mg/kg-bw for children to 1.7 mg/kg-bw for adults. Additionally, sublethal effects, including vomiting, were reported in the human incident data.

As discussed in the avian risk characterization section, the potential exposure of mammals to ethyl parathion is high because of the widespread use of ethyl parathion on several major crops. Additionally, the use of multiple applications of ethyl parathion is expected to increase the probability of exposure to mammals. Mammalian exposure to ethyl parathion can be through direct oral ingestion, dermal adsorption, and inhalation. Dermal adsorption, is likely an important route of exposure because mammals are in direct contact with contaminated foliage and soil. Small mammals, such as meadow voles or field mice, live in and around the treated fields and are particularly vulnerable because they are not expected to range far beyond the treated field. Additionally, mammals have bare skin (*e.g.*, nose and feet) areas susceptible to dermal absorption. Young mammals are expected to be at greater exposure risk than adults for the following reasons: 1.) they

consume more than adults and 2.) they generally have less hair than adults (Atterberry et al. 1997). Also, there are incomplete detoxification enzyme systems in young.

### iii. Aquatic Organisms Risk Characterization

It is likely that ethyl parathion use can adversely effect aquatic organisms on both an acute and chronic basis. Chronic RQs are extremely high for both freshwater and estuarine/marine and invertebrates.

A 100-foot buffer is required between treated areas and bodies of water. The risk assessment considers the buffer zone only for contributions to the aquatic EECs due to spray drift. Currently available methods do not enable a quantitative assessment of the mitigatory effect of buffer zones for runoff, although the buffer is likely to result in reduced contribution of runoff to aquatic EECs. The effect of the buffer may slightly reduce acute risk to fish. However, the large magnitude of exceedences for chronic risk to fish and acute and chronic risk to aquatic invertebrates suggests that the buffer is not likely to reduce the Agency's concerns for these aquatic risks.

#### Freshwater Fish

PRZM/EXAMS EECs indicate that all but the lowest label rate (alfalfa 0.5 lbs a.i./A) result in exposure to freshwater fish above acute LOC. The RQ for alfalfa was 0.3, which exceeds the restricted use (0.1) and endangered species (0.05) LOCs. The acute RQ for the other crops which have PRZM-EXAMS scenarios (corn, cotton, sorghum, soybeans) are much higher; all exceed the high risk LOC (0.5). The highest acute RQ (3.4 or 6.8 times the LOC) was for sorghum.. The RQs analysis for chronic exposure show increased risk over the acute analysis. The chronic RQs for alfalfa, corn, cotton, sorghum, and soybeans were, 9.4 (8 applications), 2.4 (2 applications), 78.8, 118.8, 131.1 and 27.6, respectively. Because a 100 foot buffer is required for ethyl parathion, additional PRZM-EXAMS modeling was conducted to assess the impact of the buffer strip on spray drift mitigation. Based on the modified spray drift assessment, the 100 feet buffer reduces the median drift from the 5% of applied default value to 2% of applied. This reduction in drift reduces the estimated acute environmental concentrations (EECs) by approximately 10%, which does not alter conclusions in the risk assessment for fish. The effect of the 100 foot buffer on runoff from the treated area is not currently known, although it is likely to reduce aquatic EECs. This adds uncertainty to risk conclusions for fish.

Open literature studies on methyl parathion cited in the risk assessment suggest that exposure to ethyl parathion may cause sublethal effects on freshwater fish. These effects as described by Henry (1984, MRID No.:44371607) include behavioral changes that result in:

- ! Lower survival and reproduction,
- ! Reduce growth due to damaged food supply,
- ! Indirect mortality

These effects are expected to occur at lower concentrations than the LC50 values used to derive the RQs.

Ethyl parathion is expected to move into water bodies via spray drift and runoff. Given that ethyl parathion is used on major crops in the Great Plains region (sunflowers, oats, sorghum, soybeans, and corn) and Georgia (corn and soybeans), there is high degree of certainty that freshwater fish can be exposed to ethyl parathion. A fish kill incident from ethyl parathion use was reported in Nebraska. (See Appendix: Table 2: Fish Incidents.)

#### Freshwater Aquatic Invertebrates

Laboratory studies submitted to EPA indicate that ethyl parathion is likely to cause adverse effects in freshwater invertebrates under all labeled ethyl parathion use scenarios. The PRZM-EXAMS RQs range from 25 to 1522.5 and 350 to 18700 for acute and chronic exposure, respectively. The RQ values above exceed LOCs by at least an order of magnitude. Therefore, even considering the uncertainty of exposure estimates from PRZM-EXAMS, it is highly likely that ethyl parathion will cause adverse effects in freshwater invertebrates.

Damage to populations of freshwater aquatic invertebrates can cause additional damage to the ecosystem. In many cases, invertebrates comprise the base of the food supply for fish. Hence, removal of this trophic level can cause linked adverse effects through the food chain. An imbalance in the predator-prey relationship allows one species to overpopulate a body of water and affect the survival of many other species. For instance, Crossland (MRID 44371714) reported that adverse effects from methyl parathion on freshwater invertebrates led to an algae bloom which caused a fish kill by depleting dissolved oxygen in treated ponds.

#### Estuarine and Marine Fish

EFED concludes that ethyl parathion poses an acute and chronic risk to estuarine and marine fish for all ethyl parathion uses except acute effects from the alfalfa use. Acute estuarine and marine fish RQs exceed all LOCs for 4 crops: corn (0.75 lbs/A), sorghum (1.0 lbs/A), cotton (1.0 lbs/A), and soybean (0.75 lbs/A). Restricted use and endangered species LOCs were also exceeded by the alfalfa (0.5 lbs/A). There is greater uncertainty for these RQs than for those for freshwater fish, because PRZM and EXAMS are not designed to simulate estuarine and marine environments.

While studies were not available for ethyl parathion, open literature data show that exposure to the close chemical analog, methyl parathion, has caused adverse effects to estuarine and marine fish. For instance, a study of methyl parathion on striped bass spawn in the delta between the Sacramento and San Joaquin Rivers correlated declines in the larval bass population with the pounds of methyl parathion applied. The effects of methyl parathion exposure on estuarine and marine fish also include behavioral changes, cholinesterase inhibition, and ovarian damage.

Although ethyl parathion is predominately used in noncoastal regions, there are coastal areas in several high use areas including Texas, Florida, Louisiana, Alabama, Georgia, and Delaware. Within these regions, ethyl parathion may move into marine and estuarine environments. The use of surface water modeling to estimate concentrations in estuarine/marine systems adds uncertainty to the



analysis. A more detailed discussion of species that might be exposed to ethyl parathion in use areas can be found below.

### Estuarine and Marine Invertebrates

With the exception of molluscs, estuarine/marine invertebrates are extremely sensitive to ethyl parathion. Although there is uncertainty associated with model-generated EECs for estuarine/marine environments, the RQs so derived exceed the LOCs by as many as three orders of magnitude. Estuarine and marine invertebrates would be harmed by concentrations of ethyl parathion much lower than predicted by the models.

### Estuarine/Marine Fish and Invertebrates Likely to Be Affected

The coastal areas of the Gulf States include a vast area of wetland habitats for estuarine species. For instance, Texas has over 300,000 acres of tidal flats, the most in the nation. Tidal flats are an important habitat and feeding ground for coastal shorebirds, fish, and invertebrates such as crabs, oysters, clams, shrimp and mussels. Texas ranks second in the nation in total area of salt marshes, with about 480,000 acres, and third in the nation in freshwater marshes with approximately 530,300 acres. Freshwater marshes, which are located upstream along river valleys, support a variety of species of fish, birds, and fur-bearing animals, as well as shrimp and crayfish.

Runoff of ethyl parathion into shallow aquatic areas may cause hazardous exposure to many commercially important estuarine species. Game fish, shrimp and crabs will visit shallow water of these estuarine habitats in the late spring and summer when ethyl parathion runoff is likely. Species such as red and black drum, sea trout and blue crabs spawn in estuaries or shallow bays, and male crabs remain there after breeding. Black drum thrive in water so shallow that their backs are exposed, and red drum feed in water shallow enough that their tails emerge from the water when they feed. Other important commercial species such as yellow flounder and brown, white and pink shrimp also spend a portion of their lives in estuaries.

#### vi. Bees and Beneficial Insects Risk Characteristics

Ethyl parathion is highly toxic to bees. Incident reports confirm bee kills from ethyl parathion use on sunflowers and alfalfa/wheat. Other crops may have unreported incidents because of inconsistencies in the incident reporting system. The American Beekeeping Federation, Inc. surveyed its members to determine the extent of damage to bee colonies due to pesticide exposure. Sixty beekeepers, operating 127,950 colonies in 22 states, reported that bee losses from pesticides are a significant issue in their operations. The survey also listed the pesticides in order according to number of bee kill responses in survey as follows: Furadan, PennCap-M, Sevin, and ethyl parathion. Because ethyl parathion poses a risk to beneficial insects, EFED recommends label language to minimize adverse effects to bees.

## References

Atterberry, T.T.; Burnett, W.T.; Chambers, J.E. Age-related differences in parathion and chlorpyrifos toxicity in male rats: Target and nontarget esterase sensitivity and cytochrome P450-mediated metabolism. *Toxicology and applied Pharmacology*. Vol. 147 Issue 2.

Bennett, R.S. et al. 1990. Effects of the Duration and Timing of Dietary Methyl Parathion Exposure on Bobwhite Reproduction. *Environmental Toxicology and Chemistry*, Vol. 9, pp. 1473-1480 (MRID No.:44371608)

Bennett, R.S. and B. A. Williams. 1991. Effects of Dietary Exposure to Methyl Parathion on Egg Laying and Incubation in Mallards. *Environmental Toxicology and Chemistry*, Vol 10, pp.501-507. (MRID No.:44371602)

Bird, S.L., Easterly, D.M., and Perry, S.G. J. *Environ. Qual.* 25 (1996).

Blondell, J.M., 1998. personal communication. USEPA. Health Effects Division.

Brewer, L.W. et al. 1988. Effects of Methyl Parathion in Ducks and Duck broods, *Environmental Toxicology and Chemistry*, Vol. 7., pp. 375-379. (MRID No.:44371604)

Brooks, H.L. et al. 1973. Insecticides, Cooperative Extension Service, Kansas State University, Manhattan, Kansas.

Buchel, K.H., 1983. *Chemistry of Pesticides. Agents for Control of Animal Pests - Organophosphates Insecticides*. Wiley-Interscience.

Buerger, T.T. 1991. Effects of Methyl parathion on Northern Bobwhite Survivability. *Environmental Toxicology and Chemistry*, Vol. 10, 527-532. (MRID No.: 44371606)

California Department of Food and Agriculture. 1987. Incidence of Multiple Case Systemic Illnesses of Agricultural Field Workers from Exposure to residues of Organophosphate Pesticides in California, 1949 through 1986. Worker Health and Safety Branch. Division of Pest Management, Environmental and worker Safety. 1220 N Street, Sacramento, California

Crossland, N.O. 1984. Fate and Biological Effects of Methyl Parathion in Outdoor Ponds and Laboratory Aquaria. *Ecotoxicology and Environmental Safety* 8, 482-495.

Dunning, J.B. Jr. 1984. Body Weights of 686 Species of North American Birds, Western Bird Banding Association, Monograph No. 1.

Fite, E. 1995. Draft of Mammalian Risk Assessments. EEB/EFED/U.S.EPA.

Fletcher, J.S., Nellessen, J.E., and Pfleeger, T.G. 1994. Literature Review and Evaluation of the EPA Food-chain (Kenaga) Nomogram, An Instrument for Estimating Pesticide Residues on Plants, *Environmental Toxicology and Chemistry*, Vol. 13, No.9, pp. 1383-1391.

Fleming, W.J. 1981. Recovery of Cholinesterase Activity in Mallard Ducklings Administered Organophosphorus Pesticides. *Journal of Toxicology and Environmental Health*, 8:885-897. MRID No.: 444489801

Fleming, W. J., 1982. Parathion Accumulation in Crickets Frogs and Its Effect on American Kestrels. *Journal of Toxicology and Environmental Health*, 10:921-927. MRID No.: 44338805.

Gordon, J.J., Leadbeater, and Maidment, M.P. 1978. The Protection of Animals Against Organophosphate Poisoning by Pretreatment with a Carbamate, “ *Toxicology and Applied Pharmacology*, vol.43,pp.207-216

Grue, C. E., Tome, M.W., Swanson, G.A., Borwick, S.M. and DeWeese, L.R. 1988. Agricultural Chemicals and the Quality of Prairie-pothole Wetlands for Adult and Juvenile Waterfowl -What are the concerns? Pages 55-64 in P.J.Stuber (Coord.) *Proceedings National symposium on Protection of Wetlands from Agricultural Impacts*. USDI, Fish and Wildlife Service Biological Report 88(16). 221 pp.(MRID No.:44357802)

Grue, C.E. 1982. Response of Common Grackle to Dietary Concentrations for Four Organophosphate Pesticides. *Arch. Environm. Contam. Toxicol.* 11, 617-626. (MRID No.:00131892)

Gunther, F.A., *Residue Reviews* 81 (1981)

Hall, R. J., Kolbe, E., 1980. Bioconcentration of Organophosphorus Pesticides to Hazardous Levels by Amphibians. *Journal of Toxicology and Environmental Health*, 6:853-860.MRID No.: 44042901.

Hartley, D., Hamish, K., eds. 1987. *The Agrochemicals Handbook*, second edition, The Royal Society of Chemistry, The University, Nottingham NG7 2RD, England.

Hernández, D. A., Lombardo, R. J., Ferrari, L. and Tortorelli, M. C. 1990. Toxicity of Ethyl-parathion and Carbaryl on Early Development of Sea Urchin. *Bulletin of Environmental Contamination Toxicology*. 45:734-741. MRID No.: 44371708.

Hill, E. F., Heath, R.G., Spann, J. W. and Williams, J.W. 1975. Lethal Dietary Toxicities of Environmental Pollutants to Birds. USFWS. *Special Scientific Report--Wildlife* No. 191. Washington. (MRID No.: 00022923)

Howard, Philip H. *Handbook of Environmental Fate and Exposure Data for Organic Chemicals*, Volume III Pesticides, Lewis Publishers Inc.(1991).

Hudson, R.H., Tucker, R.K., Haegele, M.A. 1984. Handbook of Toxicity of Pesticides to Wildlife. 2nd Edition. USDI, FWS. Resource Publication 153. Washington, D.C.

Kenaga, E.E. 1973. Factors to be Considered in the Evaluation of the Toxicity of Pesticides to Birds in Their Environment. Environmental Quality and Safety Global Aspects of Chemistry, Toxicology and Technology as Applied to the Environment Vol. II Eds: Coulston, Albany, N.Y. Korte, F. Munich. Georg Thieme Publishers, Stuttgart Academic Press, Inc., New York, N.Y.

Larson, S.J., Capel, P.D. and Majewski, M.S., 1997. Pesticides in Surface Waters. Chelsea, MI: Ann Arbor Press, 372 pp.

Maddy, K.T., Smith, C. R., Brittain, Y. and Fredrickson, S. 1985. Summary of Field Activities Following Exposure and Illness of Grapefruit Harvest Employees in Tulare County in July 1985. California Department of Food and Agricultural. Division of Pest Management, Environmental Protection and Worker Safety. Protection and Worker Safety Branch, 1220 N Street, Sacramento, California 95814

Majewski, M. S. and Capel, P.D. Pesticides in the atmosphere, Ann Arbor Press Inc. (1995).

Martin, A.C., Zim, H. S., Nelson, A. L. 1951. American Wildlife & Plants, A guide to wildlife food habits. Dover Publications, Inc., New York.

Mineau, P. 1991. Cholinesterase-inhibiting Insecticides Their Impact on Wildlife and the Environment. Chemicals in Agriculture Volume 2. Elsevier. New York.

Morgan, D. P., 1976 Recognition and Management of Pesticide Poisonings, Fourth Edition. U.S. Environmental Protection Agency, EPA-540/9-80-005

Morgan, D. P., 1989 Recognition and Management of Pesticide Poisonings, Fourth Edition. U.S. Environmental Protection Agency, EPA-540/9-88-001

Mulla, M.S., Mian, L.S., and Kawecki, J. A. 1981. Distribution, transport, and fate of the insecticides malathion and parathion in the environment. Residue Reviews 81: 1-172.

Rattner, B.A., Sileo, L., Scanes, C.G. 1982. Oviposition and the Plasma Concentrations of LH Progesterone and Corticosterone in Bobwhite Quail (*Colinus virginianus*) fed Parathion. Journals of Reproduction & Fertility Ltd **66**, 147-155. MRID No.: 44329401

Rattner, B.A., Sileo, L., Scanes, C.G. 1982. Hormonal Responses and Tolerance to Cold of Female Quail following Parathion Ingestion. Pesticide Biochemistry and Physiology **18**, 132-138. MRID No.: 44342003

Rodriguez, E. M., Monserrat, J.M., Amin, O.A., 1992. Chronic Toxicity of Ethyl Parathion and

Isobutoxyethanol Ester of 2,4-Dichlorophenoxyacetic Acid to Estruarine Juvenile and Adult Crabs. Archives of Environmental Contamination and Toxicology. 22, 140-145.

Ronis, M.J.J., and T.M. Badger, 1995. Toxic Interactions between Fungicides that Inhibit Ergosterol Biosynthesis and Phosphorothioate Insecticides in the Male Rat and Bobwhite Quail (*Colinus virginianus*), Toxicology and Applied Pharmacology 130, 221-228 (MRID No.: 44570801)

Sauer, J. R., J.E. Hines, G. Gough, I. Thomas, B.G. Peterjohn. 1997. The North American Breeding Bird Survey Results and Analyses, Version 96.4 Patuxent Wildlife Research Center, Laurel, MD

Stansell, K. 1993. Acting Director of Ecological Services. Attachment Parathion (Pesticide profile prepared by USFWS for the Endangered Species Act Section 7 consultation with the USEPA) Letter dated 6-9-1993 to Mr. Victor Kimm, Acting Assistant Administrator, EPA.

Stavola, A. 1987. Parathion Special Review Risk Assessment, Ecological Effects Branch. Hazard Evaluation Division. US E.P.A.

Stephan, C.E. 1977. Methods for Calculating an LC50. Aquatic Toxicology and Hazard Evaluation, ASTM STP 634, F.L. Mayer and J.L. Hamellink, Eds., American Society for Testing and Materials, pp. 65-84.

White, D.H., Mitchell, C. A., Hill, E. F. 1983. Parathion Alters Incubation Behavior of Laughing Gulls. Bulletin Environmental Contamination Toxicology. 31,93-97

Wolfe, C. W., Baxter, W.L., Munson, J.D., 1971. Effects of Parathion on Young Pheasants. University of Nebraska. Quarterly, Summer, vol. XVIII

USDI. 1951. Food of Game Ducks in the United States and Canada. Research Report 30. Reprint of USDA Technical Bulletin 634-1939.

USEPA. 1991. Parathion: Intent to Cancel and Deny all Registrations for Pesticide Products Containing (Ethyl Parathion). Special Review and Reregistration Division (H7508W), Office of Pesticide Programs.

USEPA. 1996. Draft Corn Insecticide Cluster Analysis. Environmental and Fate Effects Division. Office of Pesticide Programs.

USEPA. 1993. Wildlife Exposure Factors Handbook. Office of Health and environmental Assessment. Office of Research and Development. EPA/600/R-93/187b.

Cheminova. 1997. Ethyl Parathion SMART Meeting













Date	Species	Number	Crop	St	Residue Analysis		ChE
10-77	Canada, Snow, White front geese	killed/ 79 ?	wheat	OK	2 geese	12 and 14.5	None
		wheat			29 to 89		
		27					
		TOTAL 106					
Wheat field on wildlife refuge was mistakenly sprayed with EP. when neighboring wheat was treated for a greenbug infestation. Wheat for residue testing was collected 21-hr post-spray and post-mortality. (ODWC)							
6-27-78	chickens	killed / 9	cotton	TX	GI tracts		
Tests were positive for EP but values are not reported (USFWS).							
6-28-78	laughing gulls	killed / >216	cotton	TX	GI tracts	0.02-10 ppm	57-90%
Adult birds and chicks were found dead up to 3 miles from the field where EP was used at 1 lb/A. It is believed that adults feeding chicks EP contaminated insects resulted in their deaths (USFWS).							
3-14-79	Red winged blackbird, Common grackles	Killed/ 5,120	corn	NY	Red winged blackbird gizzard	617 and 1,112	none
					Grackle gizzard	150	
					Brownheaded cowbirds gizzard	171 and 166	
					American kestrel gizzard	8.9	
					corn cob	33457	

Date	Species	Number	Crop	St	Residue Analysis		ChE
Misuse corn had been treated with EP to kill birds. (Stone, 1984)							
3-20-80	ducks (8), turkeys (6), pheasants (12), chicken (1)	killed/27	wheat	OK	none	none	none
These birds were reported after the application of EP To wheat(Logan. OK) (ODWC)							
12-80	Canada geese	killed/500	winter wheat	TX	Proventricular contents	6-20 ppm	77.15
The geese were discovered around a playa lake near Etter, TX (Stavola,1987)							
1-81	geese (1500), ducks (100)	1600	wheat	TX	bolusa of wheat		none
					EP	17	
					Methyl parathion	6.1	
The birds were discovered at a playa lake near Etter, TX (Stavola, 1987)							
2-25-81	Canada geese (60), snow geese (6), white fronted geese(4), Ross's geese (2)	killed	winter wheat	TX	proventricular contents	6-20 ppm	78-85%



Date	Species	Number	Crop	St	Residue Analysis		ChE
3-30-82	Red-black-bird, Common grackle , Mournin g dove Cooper' s hawk, Red tailed hawk, Blue jay, Europea n starlin g, Eastern meadowl ark, Brownhe aded cowbird , song sparrow	killed/ 3,196	rye	NY	none	none	none
Found dead in fields containing scattered rye seeds treated with EP. (USFWS-P)							
8-82	Red-winged black-bird, Common grackle	killed/ ?	corn	PA	none	none	none











Date	Species	Number	Crop	St	Residue Analysis		ChE
8-26-89	Canada geese, Gull	kill/7	?	SD	stomach	125 ppm	
Birds were found near the Clear Lake airport (SDDA).							
1-19-90	Canada geese	killed/6 effected/ 4	small grain (wheat or barley)	WA	stomach contents	positive for EP	92%
Refuge (Toppenish Nat'l Wildlife Refuge) personnel have not been able to locate anyone who was spraying in the immediate area during January. (USFWS)							
4-4-90	blackbirds	kill/30	Corn	NC			
Farmer intentionally poisoned corn in his field to protect crop from birds. Endosulfan was also in the formulation used (NCDA).							
6-1-90	purple martin	kill/15	grain	DE			
EP was applied aerially to grain to control army worm (DEDA).							
6-11-90	starling, grosbeak, swallow, eastern bluebird	kill/5	wheat	DE			
Treatment was for saw fly in a residential wheat field. Aerial application in Sussex county (DEDA).							
7-12-90	swallows	6	barley	ND	EP whole bird	0.65 µg/g-	-

Date	Species	Number	Crop	St	Residue Analysis		ChE
					Methyl parathio n whole bird	0.043 μg/g	
Spray drift from a 0.75 to 1 Lbs a.i./A application to barley for treatment of armyworms resulted in the death of the swallows. The birds were nesting above the doorway of the house. When the home owner notice saw spray drift coming through the trees at the edge of her property. Product was Clean Crop 6-3 Parathion- Methyl Parathion. Fined for applying a pesticide not in accordance with the label causing potential human injury ... (NDDA)							
9-90	Purple martins	killed/ 15-20	small grain	DE	not detected	none	none
Lady found dead birds in her yard after an field 200 yds away was sprayed with EP 8E at a rate of 8 oz/A. The state veterinarian conducted an autopsy on 1 of the birds and felt that a toxin was involved. (DEDA)							
1990	Starlin g, Barn Swallow s, Blue Gross- beak, Bluebir d	killed/ 2  1  1 1	small grain	DE	none	none	none
Found in yard near field which was sprayed at a rate of 3/4 pints/A of Parathion 8E. (DEDA)							
5/26/ 92	Prairie Chicken s	3	Winter Wheat	MO	-	-	Level was 9.5 but no contr ols were avail able

Date	Species	Number	Crop	St	Residue Analysis	ChE
Paraspray 6-3 was aerially applied to winter wheat at 0.75 Pt a.i./A to kill armyworms. (MDA)						
3/27/93	Canada geese, Mallards, Teal	53 12 4	Corn, Winter wheat	WV	stomach contents of mallard and Canada goose  stomach contents of 2 Canada geese	75.5  41.7  Results not available
The University of Georgia, Southeast Cooperative Wildlife Disease Study indicate a final diagnosis of EP toxicosis. The crop contained some cracked corn. Gizzard was filled with grit and dark brown to green ground plant material.(SCWDS)						

## References:

- (CDFG) California Department of Fish and Game  
 (DEDA) Delaware Department of Agriculture, Division of Consumer Protection  
 (ODWC) Oklahoma Department of Wildlife Conservation  
 (MDA) Missouri Dept. of Agriculture, Bureau of Pesticide Control  
 (NDDA) North Dakota, Department of Agriculture  
 (NYSDEC) New York State Department of Environmental Conservation  
 (SCWDS) The University of Georgia, Southeastern Cooperative Wildlife Disease Study  
 (USFWS) United States Fish and Wildlife Service  
 (USFWS-P) United States Fish and Wildlife Service, Patuxent Wildlife Research Center, Laurel, Maryland  
 (VADGIF) Virginia Department of Game and Inland Fisheries

### Table 2: Aquatic Species Incidents

No./ Date	Species	Effect/ #	Crop	St	Pesticides	Residue Analysis		ChE
						Item	Conc. (ppb)	(%)
B0000-216-21  6/03/77-6/06/77	Fish (species not reported)	>1000	Rice (note: no longer a registered use)	CA	ethyl parathion	Ricefield Water	1.0	Not reported
Incident report from California Department of Fish and Game								
B0000-400-01 and I000598-008 6/27/86	Fish (bass, catfish, and minnows)	two dozen	Not reported (note: incident references adjacent registered aerial applications of ethyl parathion to fields)	NE	ethyl parathion	Not reported	Not reported	Not reported
Incident report from Nebraska Environmental Control lists origin of fish kill as "probable runoff of parathion from aerial spraying." The incident report does not include a discussion of the crop to which ethyl parathion was applied.								

Table 3: Bee Kills

Date	Crop	St	Conc. (ppm)
8-13-83	sunflower	SD	0.14-0.23
It was decided that the beekeeper had been given adequate warning of the spraying but failed to move his hives to a safe distance. Sunflowers were aerial treated with EP at a rate of 3/4 lbs/A (SDDA)			
8-24-83	sunflower	SD	bees and soil: 0.0013
The bee keeper was in the process of removing hives from next to the field as the field was treated. It is likely that he was exposed in the process.			
8-14-84	sunflower	SD	0.98
Sunflower were aerial treated with EP at 1 lbs/A to control seed weevils. Bee yard was approximately 200 yards from the sunflower field. Incident occurred near Oldham. Three hives were affected. (SDDA)			
8-18-84	sunflower	SD	none
Sunflowers 3/4 of a mile from the hives were treated with parathion. (SDDA)			
9-1-84	sunflower	SD	4.56
The incident occurred 2 mi south of Roslyn. Parathion was applied aerially and bees were found dead in front of their hive.			
6-22-85	alfalfa-wheat	SD	soil: 0.07
Winner SD. Parathion applied for grasshopper control. Soil in the bee yard had concentrations of 0.07 ppm. (SDDA)			
7-30-88	sunflower	SD	bee: 1.04
EP was sprayed on sunflowers at a rate of 1 lb/A. The allegedly affected hives were 3/4 to 1 1/4 miles from the hives. (SDDA)			

Table 4: Birds Observed in Corn Field Studies

Number	Bird	Carbofuran				Terbufos	Phorate
		FL	IL	IA	TX	MD	MD
1	American Avocet				X		
2	American Coot				X		
3	American Crow			X			
4	American Goldfinch		X	X		X	X
5	American Kestrel	X			X	X	
6	American Redstart					X	X
7	American Robin		X	X		X	X
8	American White Pelican	X					
9	Bald Eagle	X				X	X
10	Bank Swallow		X	X	X	X	
11	Barn Owl	X					
12	Barn Swallow	X			X	X	X
13	Black and White Warbler					X	
14	Black-bellied Plover	X					
15	Black-bellied Whistling-Duck				X		
16	Black-capped Chickadee		X				
17	Black-crown Night Heron	X			X		
18	Black-necked Stilt	X			X		
19	Black-shoulder Kite				X		
20	Black Tern	X					
21	Black-throated Green					X	X



Number	Bird	Carbofuran				Terbufos	Phorate
		FL	IL	IA	TX	MD	MD
22	Black Vulture					X	X
23	Blue-gray Gnatcatcher				X		
24	Blue Grosbeak				X	X	X
25	Blue Jay		X	X		X	X
26	Blue-winged Teal	X			X		
27	Blue-winged Warbler					X	X
28	Bobolink		X				
29	Bronzed Cowbird				X		
30	Brown-crested Flycatcher				X		
31	Brown-headed Cowbird		X	X	X	X	X
32	Brown Pelican	X					
33	Brown Thrasher		X	X	X	X	X
34	Buff-bellied Humming Bird				X		
35	Buff-breasted Sandpiper	X					
36	Canada Goose				X	X	X
37	Canada Warbler				X		
38	Cedar Waxwing		X			X	X
39	Carolina Wren		X			X	X
40	Caspian Tern	X					
41	Cattle Egret	X				X	X
42	Chickadee spp.					X	X
43	Chimney Swift					X	X
44	Chipping Sparrow			X		X	X
45	Cliff Swallow		X		X	X	

Number	Bird	Carbofuran				Terbufos	Phorate
		FL	IL	IA	TX	MD	MD
46	Common Crow		X				
47	Common Flicker					X	X
48	Common Grackle		X	X		X	X
49	Common Ground-dove				X		
50	Common Loon	X					
51	Common Moorhen	X					
52	Common Nighthawk		X				
53	Common Snipe	X			X		
54	Common Yellowthroat		X	X	X	X	X
55	Couch's Kingbird				X		
56	Crow spp.					X	X
57	Curve-billed Thrasher				X		
58	Dickcissel		X	X	X		
59	Double-crested Cormorant						
60	Dowitcher spp.	X					
61	Downy Woodpecker		X			X	X
62	Eastern Kingbird		X	X	X	X	X
63	Eastern Bluebird		X			X	X
64	Eastern Meadowlark		X		X	X	X
65	Eastern Pewee					X	
66	Eastern Phoebe		X		X		
67	Eastern Screech Owl	X					
68	Eastern Wood-pewee		X		X		X
69	Eurasian Tree Sparrow		X				
70	European Starling		X	X		X	X

Number	Bird	Carbofuran				Terbufos	Phorate
		FL	IL	IA	TX	MD	MD
71	Empidonax Flycatchers				X		
72	Field Sparrow		X	X		X	
73	Fish Crow	X					
74	Forster's Tern	X			X		
75	Fulvous Whistling-duck	X					
76	Glossy Ibis	X					
77	Grackle sp.					X	
78	Grasshopper Sparrow			X		X	
79	Gray Catbird		X	X		X	X
80	Gray Partridge			X			
81	Great Blue Heron	X			X	X	X
82	Great Crested Flycatcher		X	X		X	X
83	Great Egret	X			X	X	X
84	Green Heron		X		X	X	X
85	Green-back Heron	X					
86	Great-tailed Grackle				X		
87	Greater Yellowlegs	X			X		
88	Great Kiskadee		X		X		
89	Gull sp.						X
90	Hairy Woodpecker					X	X
91	Herring Gull	X				X	
92	Hooded Warbler						X
93	Horned Lark		X	X	X	X	
94	House Sparrow		X	X	X	X	X
95	House Wren		X		X	X	

Number	Bird	Carbofuran				Terbufos	Phorate
		FL	IL	IA	TX	MD	MD
96	Inca Dove				X		
97	Indigo Bunting		X	X	X	X	X
98	Kentucky Warbler				X		
99	Killdeer	X	X	X	X	X	
100	Ladder-backed Woodpecker				X		
101	Lark Sparrow		X		X		
102	Lapland Longspur			X			
103	Laughing Gull	X			X	X	
104	Least Flycatcher					X	
105	Least Sandpiper	X			X		
106	Least Tern	X					
107	Lesser Golden Plover			X			
108	Lesser Yellowlegs	X			X		
109	Lincoln Sparrow				X		
110	Little Blue Heron	X				X	X
111	Long-billed Curlew				X		
112	Long-billed Dowitcher				X		
113	Long-billed Thrasher				X		
114	Loggerhead Shrike				X		
115	Magnolia Warbler					X	X
116	Mallard	X				X	X
117	Marsh Wren						X
118	Merlin				X		
119	Mottled Duck	X			X		
120	Mourning Dove				X	X	X

Number	Bird	Carbofuran				Terbufos	Phorate
		FL	IL	IA	TX	MD	MD
121	Mute Swan					X	X
122	Myiarchas Flycatcher				X		
123	Nashville Warbler				X		
124	Northern Bobwhite Quail		X		X	X	X
125	Northern Cardinal		X		X	X	X
126	Northern Dove		X	X			
127	Northern Flicker		X	X			
128	Northern Harrier	X			X		
129	Northern Oriole		X			X	X
130	Northern Mockingbird				X	X	X
131	Northern Parula Warbler					X	X
132	Northern Pintail	X			X		
133	Northern Rough-winged Swallow				X		
134	Olive Sparrow				X		
135	Orchard Oriole		X			X	X
136	Osprey	X				X	X
137	Ovenbird					X	
138	Peafowl					X	X
139	Pectoral Sandpiper	X					
140	Pine warbler					X	X
141	Pileated Woodpecker		X				
142	Prairie Warbler						X
143	Prothonotary Warbler					X	

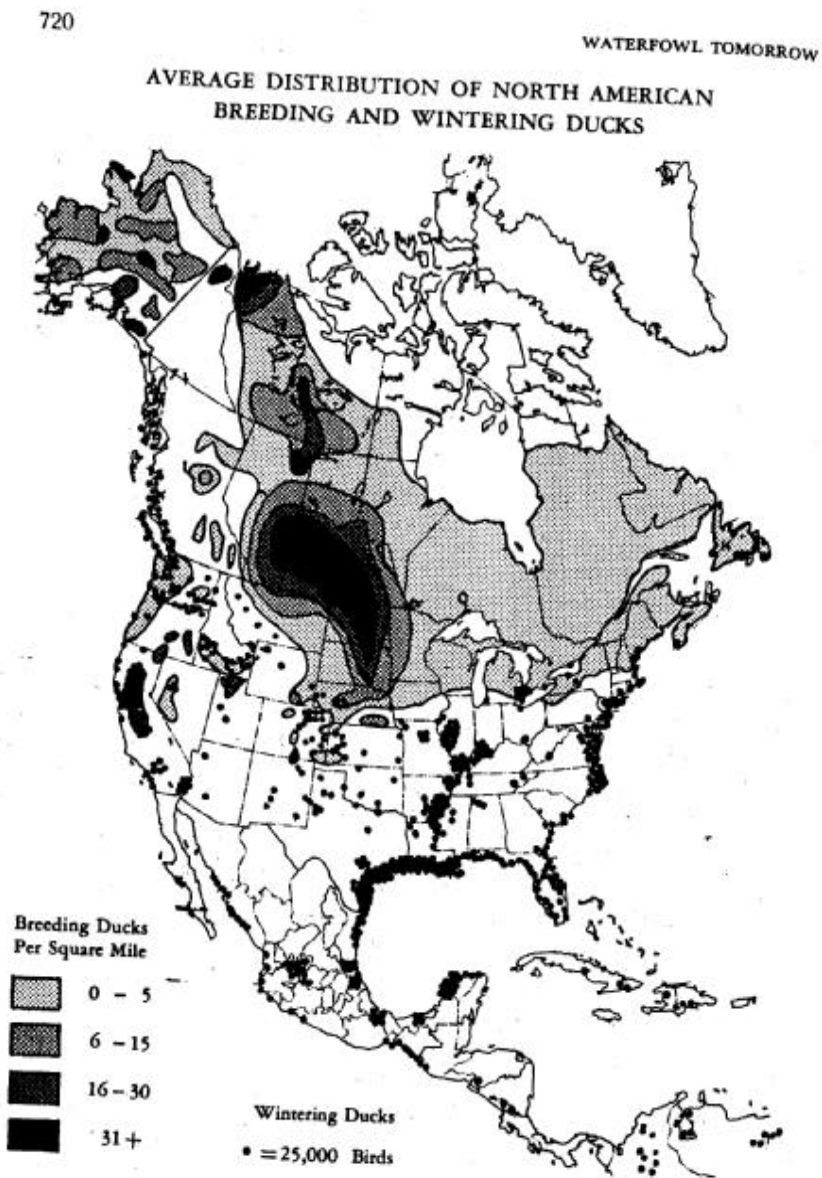
Number	Bird	Carbofuran				Terbufos	Phorate
		FL	IL	IA	TX	MD	MD
144	Purple Martin					X	X
145	Ray-breasted Warbler					X	
146	Ring-billed Gull				X		
147	Red-eyed Vireo					X	X
148	Red-bellied Woodpecker					X	X
149	Red-breasted Grosbeak		X				
150	Red-headed Woodpecker		X			X	X
151	Red-shouldered Hawk	X					
152	Red-tailed Hawk	X	X			X	X
153	Red-winged Blackbird		X	X	X	X	X
154	Ring-billed Gull	X					
155	Ring-necked Pheasant		X				
156	Rock Dove		X	X	X	X	X
157	Rose-breasted Grosbeak					X	
158	Ruby-crowned Kinglet			X	X		
159	Ruby-throated Hummingbird					X	X
160	Ruddy Duck	X					
161	Ruddy Turnstone	X					
162	Rufus-sided Towhee					X	X
163	Savannah Sparrow		X	X	X	X	
164	Sharp-shinned Hawk	X					
165	Scarlet Tanager					X	X

Number	Bird	Carbofuran				Terbufos	Phorate
		FL	IL	IA	TX	MD	MD
166	Scissor-tailed Flycatcher				X		
167	Sedge Wren			X		X	
168	Semipalmated Plover	X					
169	Semipalmated Sandpiper	X					
170	Solitary Sandpiper	X					
171	Snowy Egret	X			X	X	X
172	Song Sparrow		X	X		X	
173	Stilt Sandpiper	X					
174	Swainson's Hawk				X		
175	Tennessee Warbler		X				
176	Tree Swallow		X		X	X	X
177	Tricolor Heron	X			X		
178	Tropical Kingbird				X		
179	Tufted Titmouse		X		X	X	X
180	Turkey Vulture	X				X	X
181	Upland Sandpiper	X			X		
182	Vesper Sparrow		X	X			
183	Western Kingbird				X		
184	Western Meadowlark			X			
185	Western Sandpiper				X		
186	White Breasted Nuthatch		X				
187	White-crowned Sparrow		X				
188	White-eyed vireo					X	X

Number	Bird	Carbofuran				Terbufos	Phorate
		FL	IL	IA	TX	MD	MD
189	White-faced Ibis	X			X		
190	White-tipped Dove				X		
191	White-throated Sparrow					X	X
192	White-winged Dove				X		
193	Willet	X					
194	Willow Flycatcher		X				
195	Wilson's Phalarope	X					
196	Wilson's Warbler				X		
197	Wood Duck	X					
198	Wood Stork	X			X		
199	Wood Thrush					X	X
200	Worm-eating warbler					X	
201	Yellow-billed cuckoo					X	X
202	Yellow-breasted Chat		X		X	X	X
203	Yellow-headed Blackbird				X		
204	Yellow-rumped warbler					X	
205	Yellow Warbler		X		X	X	X



Figure 1



Chemical No: 057501

**DATA REQUIREMENTS FOR  
Parathion(ethyl)**



Data Requirement	Use Pattern <sup>1</sup>	Does EPA Have Data To Satisfy This Requirement? (Yes, No, or Partially)	Bibliographic Citation	Must Additional Data Be Submitted Under FIFRA 3(c)(2)(B)?
§158.490 WILDLIFE AND AQUATIC ORGANISMS				
71-1(a) Acute Avian Oral, Quail/Duck	1,2	Yes	00160000 1115195	No
71-2(a) Acute Avian Diet, Quail	1,2	Yes	00022923	No
71-2(b) Acute Avian Diet, Duck	1,2	Yes	00022923	No
71-3 Wild Mammal Toxicity				
71-4(a) Avian Reproduction Quail		Yes	41133102	No
71-4(b) Avian Reproduction Duck		Yes	41133101	No
71-5(a) Simulated Terrestrial Field Study				
71-5(b) Actual Terrestrial Field Study				
72-1(a) Acute Fish Toxicity Bluegill	1,2	Yes	40098001 40094602 40644710 00035796 00057051	No
72-1(b) Acute Fish Toxicity (TEP)				
72-1(c) Acute Fish Toxicity Rainbow Trout	1,2	Yes	40094602	No
72-1(d) Acute Fish Toxicity Rainbow				

Data Requirement		Use Pattern <sup>1</sup>	Does EPA Have Data To Satisfy This Requirement? (Yes, No, or Partially)	Bibliographic Citation	Must Additional Data Be Submitted Under FIFRA 3(c)(2)(B)?
72-2(a)	Acute Aquatic Invertebrate	1,2	Yes	40089001 40094602 40644711	No
72-2(b)	Acute Aquatic Invertebrate (TEP)	1,2	No	---	No
72-3(a)	Acute Est/Mar Toxicity Fish	1,2	Yes	40228401	No
72-3(b)	Acute Est/Mar Toxicity Mollusk	1,2	Yes	41237807	No
72-3(c)	Acute Est/Mar Toxicity Shrimp	1,2	Yes	40644714 01237807	No
72-3(d)	Acute Est/Mar Toxicity Fish (TEP)	1,2	No	---	No
72-3(e)	Acute Est/Mar Toxicity Mollusk (TEP)	1,2	No	40644717	No
72-3(f)	Acute Est/Mar Toxicity Shrimp (TEP)	1,2	No	40644715	No
72-4(a)	Early Life Stage Fish	1,2	No	---	No
72-4(a)	Early Life-Stage Fish (Estuarine / Marine species)	1,2	No	41543101	No
72-4(a)	Early Life Stage Fish				
72-4(b)	Life Cycle Aquatic Invertebrate	1,2	No	43583501 40874401	No
72-5	Life Cycle Fish	1,2	No	---	No
72-6	Aquatic Organism Accumulation	1,2	No	---	No
72-7(1)	Simulated Aquatic Field Study	1,2	No	---	No
72-7(b)	Actual Aquatic Field Study	1,2	No	---	No
§158.540 PLANT PROTECTION					

Data Requirement	Use Pattern <sup>1</sup>	Does EPA Have Data To Satisfy This Requirement? (Yes, No, or Partially)	Bibliographic Citation	Must Additional Data Be Submitted Under FIFRA 3(c)(2)(B)?
122-1(a) Seed Germ./Seedling Emerg.	1,2	No	---	Yes
122-1(b) Vegetative Vigor	1,2	No	---	Yes
122-2 Aquatic Plant Growth	1,2	No	---	Yes
123-1(a) Seed Germ./Seedling Emerg.	1,2	No	---	Reserved <sup>2</sup>
123-1(b) Vegetative Vigor	1,2	No	---	Reserved <sup>2</sup>
123-2 Aquatic Plant Growth	1,2	No	---	Reserved <sup>2</sup>
124-1 Terrestrial Field Study	1,2	No	---	No
124-2 Aquatic Field Study	1,2	No	---	No
§158.490 NONTARGET INSECT TESTING				
141-1 Honey Bee Acute Contact	1,2	Yes	00036935	No
141-2 Honey Bee Residue on Foliage	1,2	Yes	00060628	No
141-5 Field Test for Pollinators	1,2	No	---	No
§158.290 ENVIRONMENTAL FATE				
<u>Degradation Studies-Lab:</u>				
161-1 Hydrolysis	1,2	Yes	40478701	No
161-2 Photo degradation In Water	1,2	Yes	40644701, 42156001	No
161-3 Photo degradation On Soil	1,2	Yes	40647702, 42025501	No
161-4 Photodegradation In Air	1,2	Yes	41126601, 42158201	No
<u>Metabolism Studies-Lab:</u>				
162-1 Aerobic Soil	1,2	Yes	41187601, 42073101	No
162-2 Anaerobic Soil	1,2	No	N/A	No

Data Requirement	Use Pattern <sup>1</sup>	Does EPA Have Data To Satisfy This Requirement? (Yes, No, or Partially)	Bibliographic Citation	Must Additional Data Be Submitted Under FIFRA 3(c)(2)(B)?
162-3 Anaerobic Aquatic	1,2	Partially	41249801, 42451001	Yes
162-4 Aerobic Aquatic	1,2	Partially	41249802, 42476901	Yes
<u>Mobility Studies:</u>				
163-1 Leaching- Adsorption/Desorp.	1,2	Partially	41076701	Yes
163-2 Volatility (Lab)	1,2	Yes	40810902	No
<u>Dissipation Studies-Field:</u>				
164-1 Soil Field Dissipation	1,2	Partially	41481101, 41187602, 41292500	Yes
165-2 Aquatic Field Dissipation	1,2	Partially	41481102, 41187603	Yes
<u>Accumulation Studies:</u>				
165-4 In Fish	1,2	Partially	40988101	Yes

Data Requirement	Use Pattern <sup>1</sup>	Does EPA Have Data To Satisfy This Requirement? (Yes, No, or Partially)	Bibliographic Citation	Must Additional Data Be Submitted Under FIFRA 3(c)(2)(B)?
<u>Ground Water Monitoring Studies:</u>				
166-1 Small-Scale Prospective	1,2	No	N/A	No
§158.440 SPRAY DRIFT				
201-1 Droplet Size Spectrum	1,2	No	N/A	Reserved
202-1 Drift Field Evaluation	1,2	No	N/A	Reserved

FOOTNOTES:

1. 1=Terrestrial Food; 2=Terrestrial Feed; 3=Terrestrial Non-Food; 4=Aquatic Food; 5=Aquatic Non-Food(Outdoor);6=Aquatic Non-Food (Industrial);7=Aquatic Non-Food (Residential);8=Greenhouse Food; 9=Greenhouse Non-Food; 10= Forestry; 11=Residential Outdoor; 12=Indoor Food; 13=Indoor Non-Food; 14=Indoor Medicinal; 15=Indoor Residential.

2. Plants studies are tiered based on the results of the first tier the second tier may be required.

Draft NOIC (Notice of Intent to Cancel) 07/17/1991